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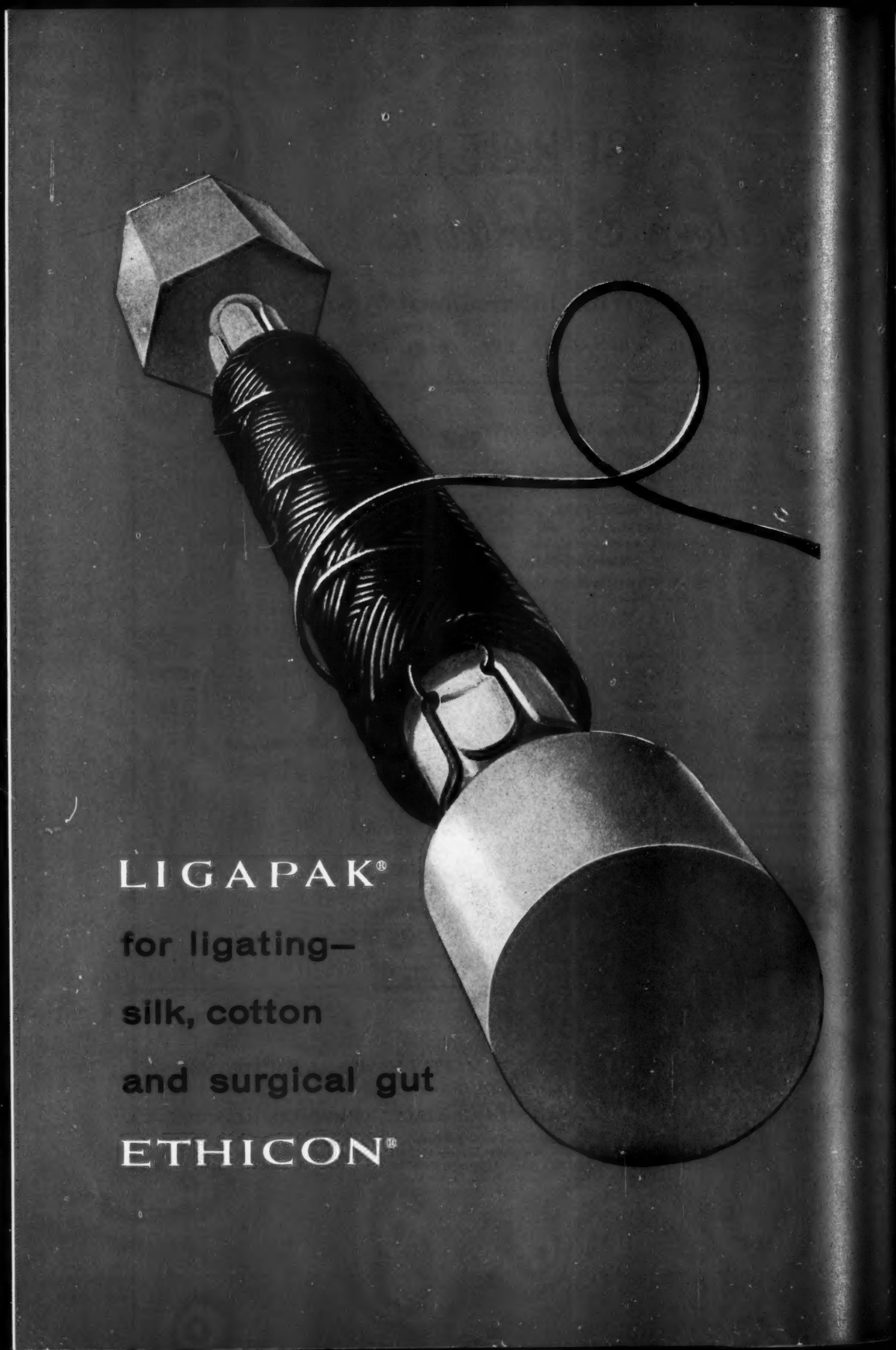
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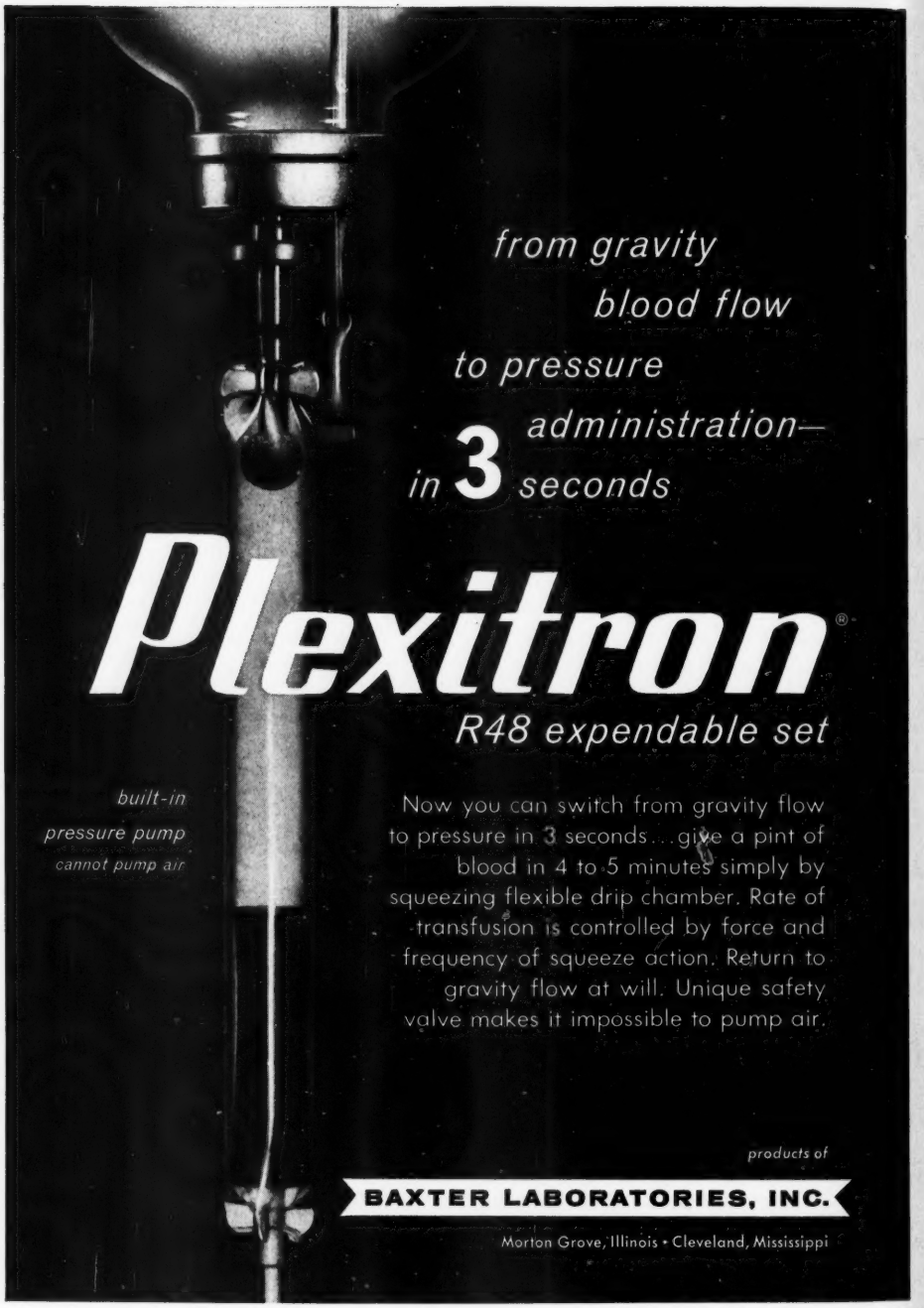
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THE BY-PASS OPERATION IN THE TREATMENT OF ARTERIOSCLEROTIC OCCLUSIVE DISEASE OF THE LOWER EXTREMITIES

E. STANLEY CRAWFORD, M.D., and MICHAEL E. DE BAKEY, M.D., Houston, Texas

DURING THE PAST FEW YEARS the surgical approach to arteriosclerotic segmental occlusive disease of the peripheral arteries has been directed toward re-establishment of blood flow through the main arterial channels. Although numerous technical variations are possible, in general three types of procedures have been employed for this purpose. These are endarterectomy, excision and graft replacement of the occluded segment, and by-pass of the diseased area with a graft.

Our experience with these methods of treatment, like that of others, has disclosed a number of factors, both technical and physiologic, that are essential to the success or failure of the operation. Most important among these are: first, the presence of an adequate patent peripheral arterial bed for the ready and unimpeded outflow of blood and, second, the performance of the procedure in a manner that will minimize tissue damage as well as interference with collateral circulation. The former can be established by proper arteriographic studies which should demonstrate not only the limits of the occlusive process but also the character of the peripheral arterial bed well beyond the distal point of the occlusion, as Lindbom has pointed out. The latter is concerned primarily with certain technical considerations that have an important bearing upon the outcome of the procedure. Operative trauma to adjacent structures, such as veins, lymphatics, and nerves, must be minimized to prevent troublesome and even incapacitating complications such as edema, neuralgia, and wound infection. Similarly, injury or damage to the main artery and to its collateral vessels, both in the region of the occlusion as well as above and below this process, must be minimized in order to prevent subsequent thrombosis. The procedure should be done in a manner that will preserve all existing collateral blood vessels in order to prevent further loss of circulation. Thus, while the procedure should be directed toward restoration of a strong pulsatile

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FIG. 1.

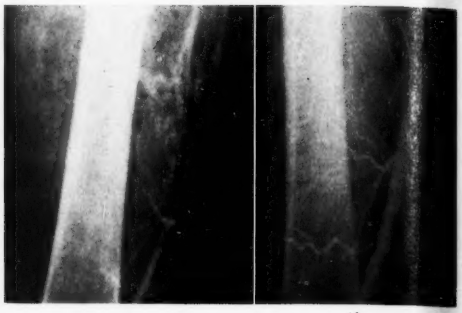

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FIG. 2.

FIG. 1. a, Preoperative arteriogram of left superficial femoral artery showing short segmental occlusion with one large and several small collateral branches. b, Postoperative arteriogram of same case showing patent graft but fewer visible collateral branches.

FIG. 2. a, Preoperative arteriogram of right superficial femoral artery showing occlusion with multiple collateral branches. b, Postoperative arteriogram of same case showing patent graft and the same collateral branches as observed preoperatively.

blood flow into the peripheral arterial bed, its performance should be associated with minimal jeopardy to the existing circulatory and functional capacity of the extremity.

The procedure of endarterectomy, except in the very short occlusive lesions, has not completely met these criteria in our experience. Postoperative thrombosis has occurred too often and at least 2 legs were made worse by the operation. Amputation was required in both cases several days later, and dissection of the limbs revealed thrombosis not only in the endarterectomized area, but also extending distally and obliterating essential collaterals. The reported experience of Bazy, Fontain and associates, and Julian and associates has been quite similar.

Complete excision with homograft replacement of an occluded segment has the disadvantages of an extensive operation, particularly in those cases with relatively long lesions. There is greater opportunity for injury to the saphenous nerve, lymphatics, and the adjacent veins. Desirable venous and arterial collateral vessels are sacrificed, thus necessitating considerable reliance on success of the operation for preservation of the extremity (Fig. 1). The extent and difficulty of such an operation, with its possible undesirable sequelae, are distinct disadvantages in those patients who are likely to have heart disease, diabetes, or cerebral arteriosclerosis.

The generally employed type of by-pass operation consists of inserting a graft by end-to-end anastomosis to the host artery above and below the diseased segment after ligating its two ends. Since this segment is often not completely occluded except for a short distance, and may still communicate with collateral vessels, blood flow through them is reduced or abolished by such interruption. Should thrombosis occur in the graft from technical errors or other complication, such as wound infection, the resulting circulation would be less than that existing preoperatively.

The type of procedure that has been found most effective in avoiding these disadvantages and in meeting the requirements described for successful results has been the bypassing operation originally described by Kunlin and his associates. It is based upon

FIG. 3.

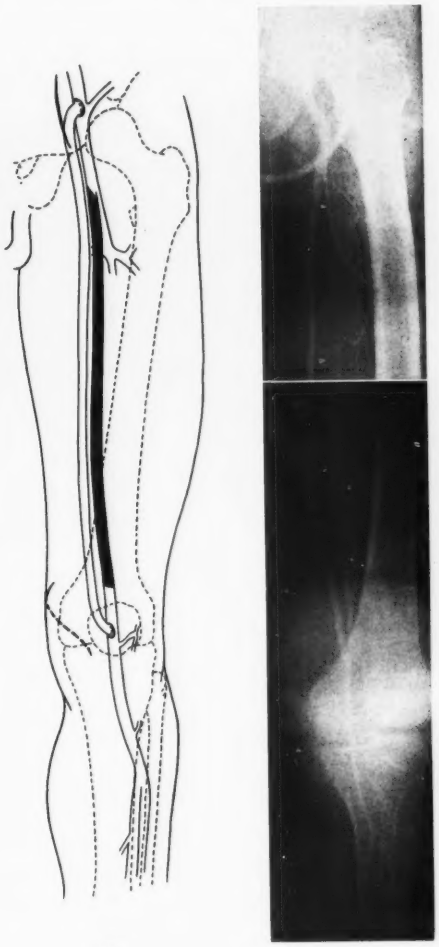

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FIG. 4.

FIG. 3. Postoperative arteriogram of right superficial femoral artery showing patent graft by-passing occlusion and not disturbing the fine arbor of collateral branches at either end of the occluded segment.

FIG. 4. a, Preoperative arteriogram of left superficial femoral artery, showing complete occlusion of the proximal half of this vessel with partial occlusion in distal half but with good lumen in popliteal artery. b, Diagram showing the segment of the superficial femoral artery to be by-passed in solid black. The arterial homograft is anastomosed to the common femoral artery above and to the popliteal artery below. c, Postoperative arteriogram showing patency of the graft and the segment of the host artery which was patent preoperatively still unchanged.

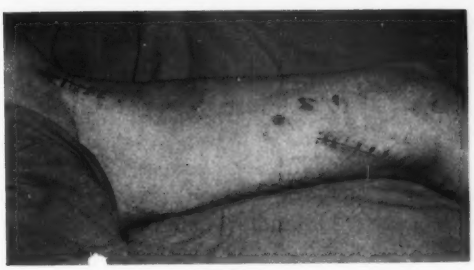

FIG. 5.

FIG. 5. The location of the two incisions used in by-passing a long occluded segment of the femoral artery such as that shown in Figure 4.

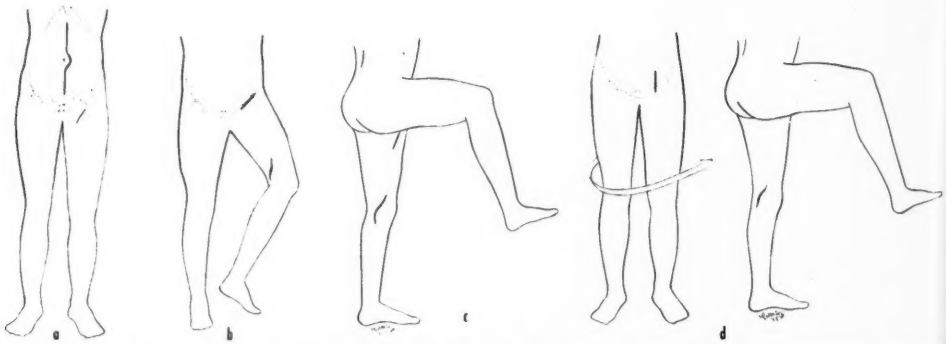

FIG. 6. Diagrams illustrating position of the patient on operating table. a, Supine position for operations to by-pass occlusions involving iliac or upper femoral arteries with midline abdominal incision to expose former artery and oblique incision in upper thigh to expose latter artery. b, Supine position with leg slightly flexed and externally rotated showing upper and lower incisions for operations to by-pass occlusions involving midportion of superficial femoral artery. c, Lateral position with uninvolved leg sharply flexed showing upper and lower incision for occlusions involving lower third of femoral artery. d, Combined supine and lateral position showing high incision to expose upper femoral artery and low incision in order to expose the popliteal artery for long occlusions which involve most of the superficial femoral artery.

the natural response of the arterial tree to an occlusion, which is the enlargement of collateral vessels around the blocked area. In essence the operation simply adds a collateral vessel about the size of the normal artery to those already present without disturbing these vital branches (Figs. 2 and 3). This is accomplished by anastomosing a graft end-to-side to the host artery above and below the diseased segment through two small separate incisions which are connected by a tunnel made with a vein stripper (Figs. 4 and 5). In our experience this method has satisfied best the criteria outlined previously, and it would seem worth while to present in detail the technique of this operation as we have used it, along with a brief analysis of our results with its application in 40 extremities, using lyophilized arterial homografts.

TECHNIQUE

The position of the patient on the operating table determines the ease of exposure, since the two incisions are often widely separated and in different planes of the body. The patient is placed in the supine position for operations to by-pass occluded segments in the iliac or upper femoral arteries (Fig. 6a). For occlusions in the midsuperficial femoral region, the same position is used, but, in addition, the involved leg is slightly flexed and externally rotated to bring the lower incision into the proper plane (Fig. 6b). When the disease is in the lower third of the superficial femoral artery, the patient lies on his side with the involved leg nearest the table and slightly flexed, while the upper or uninvolved leg is sharply flexed, thus exposing both areas to be incised (Fig. 6c). In by-passing long segments when one incision is made high in the groin or abdomen and the other behind the knee, a combination of positions may be necessary (Fig. 6d). This can be accomplished with ease by proper preparation of the skin and adequate draping.

The incisions are from 4 to 10 centimeters in length and are placed to expose the most normal area both above and below the obstruction and at a safe distance from the arbor of collaterals (Fig. 7a). The availability of the arteriogram in the operating room is essential for determining the proper placement of the incisions.

Either incision is made first, and following exposure of the artery above and below, clamps are applied on either side of the area

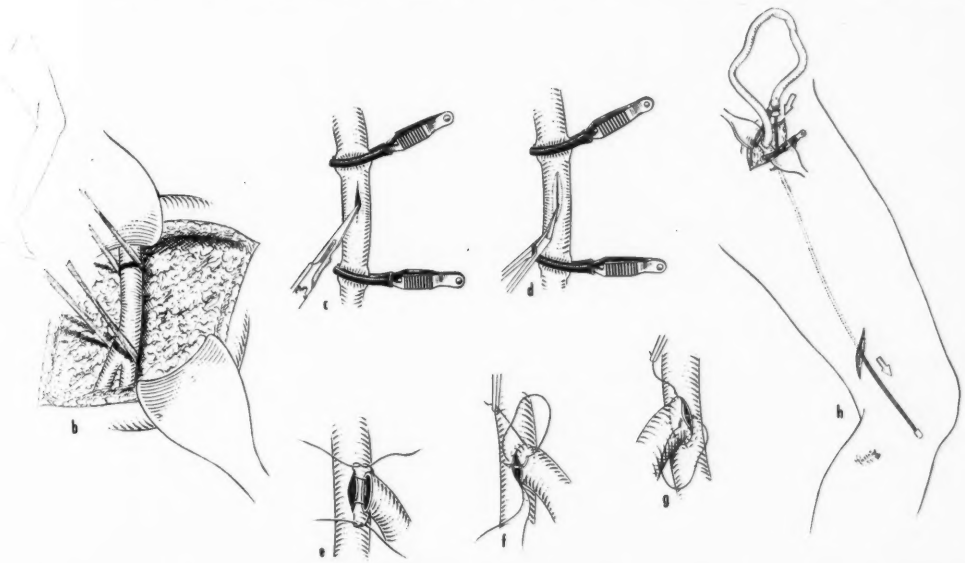

FIG. 7. Technique of performing by-pass procedure for occlusion of superficial femoral artery. a, With patient in supine position and leg slightly flexed and externally rotated upper and lower incisions are made to expose artery well above and below obstruction. b, Common femoral artery is encircled with umbilical tapes after being mobilized for a distance of about 5 centimeters, which in this instance is just above origin of profunda femoris. c, Occluding clamps are applied to the artery and a longitudinal incision about the size of the lumen of the graft is made in its wall anteriorly. d, An oval aperture is made by excising with fine scissors a small elliptic piece from the lateral edges of the wound. e, The end-to-side anastomosis between the bevelled end of the arterial homograft and the opening in the host's artery is begun with two separate sutures of No. 5-0 arterial silk which are fixed at each end of the wound. f, One suture is then used to anastomose one side as a continuous through-and-through stitch and anchored to the free end of the other suture. g, The anastomosis of the other side is done with the other suture in a similar manner. h, After this upper anastomosis is completed the other end of the graft is attached to a vein stripper and drawn through a tunnel previously made by blunt dissection into the lower incision. The procedure is then completed by an end-to-side anastomosis of this end of the graft to the artery which is exposed in the lower incision.

of artery to be incised (Fig. 7b). Ordinary bulldog clamps or other types of atraumatic arterial clamps may be used for this purpose. Following longitudinal incision of the artery a small oval aperture is created by excising with fine scissors a small elliptic piece from the lateral edges of this wound (Fig. 7c and d). The final opening is made equal in size to the greatest diameter of the bevelled end of the graft to be inserted, which in most instances is about the same as that of the host artery.

A reconstituted lyophilized arterial homograft, the ends of which have been slightly bevelled, is then sutured end-to-side to the host artery. A simple over-and-over stitch using No. 5-0 arterial silk is employed. The graft is first fixed to the patient's artery at each end of the wound by a suture that is passed from outside in and out again through the graft (Fig. 7e). Each suture is tied and the needle end of one is passed over and over from outside in through the graft, and from inside out through the host artery (Fig. 7f). The anastomosis of this side is completed by tying the suture to the short end of the other suture. Then using the second suture, the other side of the anastomosis is performed in a similar fashion (Fig. 7g). Accurate approximation is most necessary in order to prevent constriction of the patient's artery.

After the first anastomosis is completed, a tunnel connecting the two incisions is started with blunt dissection using an index finger in each wound. When the distance to be covered is long a vein stripper may be used to complete this procedure, but any long clamp will suffice for shorter distances. The free end of the graft is attached to the end of this instrument and is pulled after it to the opposite incision (Fig. 7h). Rotation or twisting of the graft must be prevented during this procedure. The second anastomosis is then performed using the same technique described before. During the second anastomosis, traction must be applied to the graft in order to attain satisfactory tension at the completion of the operation. The clamps are removed after establishing the second anastomosis and suture line bleeding is controlled by applying dry gauze for a few moments. The wounds are closed and ordinary bandages are applied.

RESULTS

Blood flow through the main arterial channel distal to an occlusion was successfully re-established in 37 of the 40 extremities in which this technique was employed. Success was determined by arteriography or by the presence of palpable pulses distal to the block that were absent prior to operation. The 3 failures were in borderline candidates evidenced by small distal patent segments with secondary occlusions below. Particularly significant is the fact that none of these cases was made worse by the operation. The clinical condition of the extremity and the postoperative arteriograms were unchanged.

The indication for operation in the 37 successful cases was intermittent claudication in 29, gangrene of limited degree in 7, and in 1, extensive gangrene. Among the patients with gangrene, painful ischemic ulcers were present in 2, severe rest pain in 6, and in 1, a digit had been destroyed by associated infection. All the patients with claudication and rest pain were relieved immediately after operation and have remained well. The ulcers in the 2 patients with this complication immediately became painless and healed rapidly. In the patient with the infected toe, amputation of this digit was followed by rapid healing of the stump. A low leg amputation leaving a long stump was successfully performed in the patient with the gangrenous foot, who also had significant lowering of cutaneous temperature at the midthigh level and color changes at the midtibial level. According to previous experience such manifestations would have necessitated a midthigh amputation. In this patient, however, there was sufficient improvement in circulation following insertion of the graft to permit amputation below the knee with a long length stump. The extra length of this stump has been of distinct advantage to this elderly patient, who had had a midthigh amputation on the other side previously.

All the grafts in this series have remained patent since discharge of the patients from the hospital and for periods up to 18 months. No patient has had a recurrence of his difficulties. There were no deaths and only a few complications. A superficial wound infection occurred in 1 patient and thrombophlebitis complicated by a small pulmonary infarct developed in another, but both patients recovered completely without residual disturbances.

SUMMARY

1. The occlusive process in chronic arteriosclerotic arterial insufficiency of the lower extremities is often segmental in nature with a patent vessel above and below it. Owing to this fortunate pathologic feature of the disease it is frequently possible to restore normal circulation in the peripheral arterial bed. This may be accomplished by several methods including thromboendarterectomy, excision of the occluded segment with replacement by a graft, and by-pass of the diseased area with a graft.

2. Our experience with these methods has led us to the conviction that the by-pass procedure, owing to its simplicity in concept

and application, is the most effective in achieving the desired objectives and is associated with the fewest disadvantages. It consists essentially in the addition of a large collateral vessel around the blocked area to those already present, but without disturbing them. Conceptually, therefore, it is based upon the natural response of the arterial tree to an occlusive process, namely, the enlargement of collateral vessels around the obstruction. The procedure is accomplished by the anastomosis of an arterial homograft end-to-side to the patient's artery above and below the occluded segment through two small separate incisions which are connected by a tunnel in the subcutaneous tissues.

The most important advantage of this procedure lies in the fact that it not only permits restoration of a normal pulsatile blood flow into the peripheral arterial bed, but also is associated with minimal jeopardy to the existing circulatory and functional capacity of the extremity. The minimal dissection required in its performance reduces the risk of injury and thrombosis to the main artery and to its collateral vessels in the region of occlusion as well as above and below this process. Because adjacent structures such as veins, lymphatics, and nerves are not disturbed, troublesome and even incapacitating complications such as edema, neuralgia, and wound infections are largely prevented.

4. These advantages are well demonstrated by the highly gratifying results obtained in our experience with the application of this procedure in 40 extremities. Restoration of a pulsatile blood flow in the peripheral arterial bed was obtained in all but 3 of the cases. These 3 failures were borderline candidates to begin with, but particularly significant is the fact that none of them was made worse by the operation.

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CONGENITAL DIAPHRAGMATIC HERNIA IN ADULTS

DAVID P. BOYD, M.D., F.A.C.S., and BART F. WOOLDRIDGE, M.D., Boston, Massachusetts

CASE REPORTS of congenital diaphragmatic hernia in the adult are rare. Herniation through the diaphragm as usually seen in adults is of the esophageal hiatus type. In infants and children, on the other hand, the commonest are those which herniate through the pleuroperitoneal hiatus or through defects produced by partial congenital absence of the diaphragm. This discrepancy is due to the high mortality produced by diaphragmatic hernias in early life.

Riverius of England, in 1698, apparently was the first to record a case of congenital diaphragmatic hernia. In 1853 Henry I. Bowditch, of Boston, was able to analyze 88 cases of diaphragmatic hernia recorded in the literature. Of these, the hernia was considered to be congenital in 26 cases. Seventeen of these 26 patients were dead within 2 years after birth. By 1922 Latta had collected 127 cases of congenital hernia due to defective formation of diaphragm. Eighty-eight of these patients were either stillborn or died within an hour or two after birth. Orr and Neff, in 1936, collected from the literature only 17 cases of congenital diaphragmatic hernia in which operation had been performed in the first year of life; 9 of these 17 patients survived. During the last few years, however, many case reports and series of congenital diaphragmatic hernia in children have been recorded and today surgical repair of all forms of diaphragmatic hernias is commonplace.

The terminology and general nomenclature of diaphragmatic hernia is somewhat cumbersome and some of the entities are ill-defined. Perhaps the complex embryologic development of the diaphragm accounts for some of this confusion. It has been emphasized many times in the past that the embryologic development of the diaphragm subjects this musculofascial structure to areas of potential weakness through which abdominal viscera may herniate into the thorax. The relative negative pressure existing in the thorax plus the added muscular pull of the diaphragm during inspiration throws a definite strain on any congenitally weak or absent areas in the organ.

The diaphragm is made up of several component parts having separate embryologic origins. These are: (1) the septum transversum, (2) the pleuroperitoneal membrane, (3) the primitive mesentery, and (4) portions of the wall of the celomic cavity. The mesenchyme of the ventral mesentery gives rise to the septum transversum. This forms a fan-like structure which is attached to the body wall ventrally and laterally, partitioning the ventral portion of the celomic cavity (Fig. 1). The dorsal portion of the cavity remains open as the paired pleuroperitoneal canals, being separated from one another by the primitive dorsal mesentery of the gastrointestinal tract. The pleuroperitoneal membranes originate from a vertical ridge of mesenchyme and grow around the lower pole of the developing lung bud to fuse with the mesonephros dorsolaterally and the mesentery medially. This fusion practically closes the pleuroperitoneal canal, leaving only a slit at its dorsomedial aspect, the pleuroperitoneal hiatus. Closure of this hiatus is finally accomplished by growth of the primitive mesentery, and the diaphragm is fully formed when the embryo has attained 20 millimeters of length (Fig.1). As growth continues, the phrenic nerve with a muscle mass descends and invades the diaphragm, partially replacing the membranous structures.

If the mesoderm is not adequately deposited at any of these points of union, or if the timing should be wrong, or if there is failure of adequate fusion, weak points in the dia-

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phragm may result. In more aggravated cases congenital continuity of the pleural and peritoneal cavities occurs. The site of occurrence of these weak areas would naturally be at the points of fusion. These points are: (1) the pleuroperitoneal hiatus, (2) the outer crus, and (3) the esophageal hiatus.

In the normal adult diaphragm there may be a triangular space of muscular deficiency between the outer crus of the lumbar portion of the diaphragm, the twelfth rib, and the costal portion of the diaphragm (Fig. 2). The apex of this vertebral-costal triangle is curved upward and forward toward the tendinous portion of the diaphragm. Its base partially rests on the twelfth rib and its lateral borders are muscular and are of a somewhat sphincterlike construction. This space is closed by an areolar membrane which consists of two sheaths; the upper sheath comes from the pleura, and the lower is a continuation of the iliac fascia. This is the space which was first described by Bochdalek and has since become known as the foramen of Bochdalek. Since this area is the last portion of the pleuroperitoneal canal to close, one might expect the commonest form of congenital diaphragmatic hernia would be through the vertebral-costal triangle. Theoretically, this could occur either as a true hernia forcing the fibrous tissue to bulge into the pleural cavity or as a prolapse of abdominal organs through the enclosed gap, forming a spurious hernia. The former type of true hernia is apparently most unusual and we know of no reported case of a hernia with a sac in this region.

Unilateral partial absence of the diaphragm probably results from failure of development of the pleuroperitoneal membrane and its fusion with the mesonephros. There is, thus, great opportunity for variation in the size and shape of the diaphragmatic deficiency. These defects are likely to be associated with extensive displacement of abdominal viscera into the thorax, producing, in turn, marked or complete collapse of the lung and mediastinal shift. Practically every structure in the abdomen has been described as having herniated into the thorax through these congenital defects but, unlike traumatic hernias, adhesions to thoracic viscera are rarely encountered.

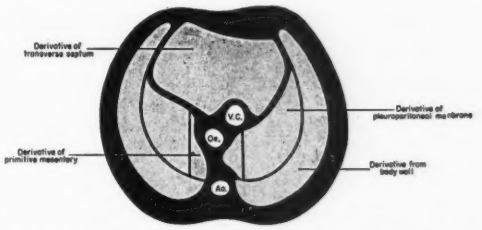

FIG. 1. This diagram illustrates the positions of the elements contributing to the developing diaphragm.

CLINICAL ASPECTS

The manifestations of these congenital hernias are protean and vary from a mild cough to the most severe interference with the function of the cardiorespiratory and gastrointestinal systems. From the literature it is clear that congenital hernias have been noticed much more frequently on the left side than on the right. Although the cause of this phenomenon is probably to be found in the embryologic formation of the diaphragm, as summarized before, it is nevertheless a subject of controversy among embryologists. It is doubtful whether the presence of the liver on the right side influences the formation of the right pleuroperitoneal membrane, but certainly it would decrease the tendency toward misplacement of viscera into the chest.

Despite the frequency of associated anomalies in other situations it is only occasionally

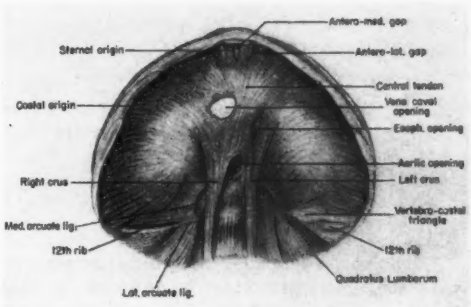

FIG. 2. The normal adult diaphragm.

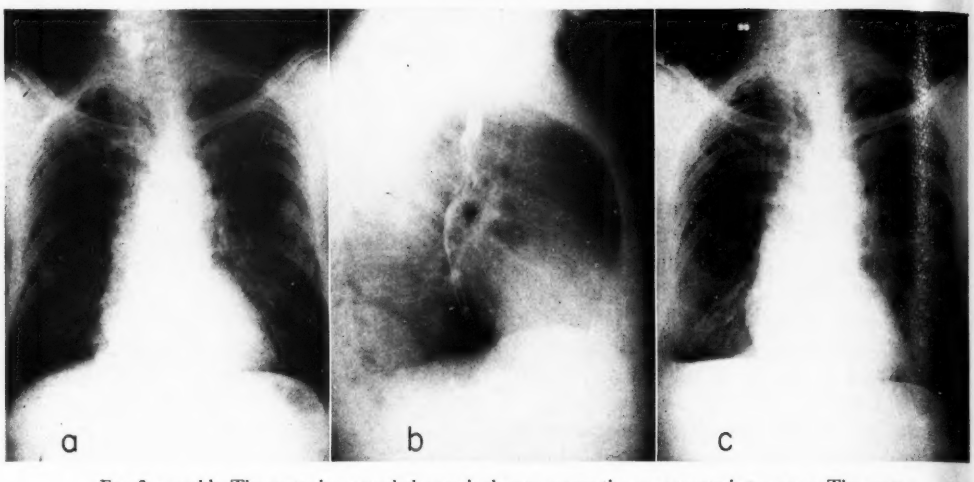

FIG. 3. a and b, The posterior rounded mass is shown, suggesting a neurogenic tumor. c, The postoperative film.

that the diaphragmatic defect is part of a multiple picture. When other defects occur they are usually underdeveloped lungs and malrotation of the bowel.

These hernias should be repaired as soon as the diagnosis has been established in order to alleviate the associated cardiorespiratory embarrassment. Larger hernias in infants probably should be approached through the abdomen, but ordinarily hernias in these cases are best repaired transthoracically. In many cases repair of the defect consists of placement of just a few sutures, but in other cases reduction of the large hernia into the abdomen may present a serious problem. The use of the relaxing agent anectine has proved of aid to us in this situation. At times phrenic nerve crushing, utilization of the perirenal fascia or latissimus dorsi muscles, or lower rib thoracoplasty may be necessary to obliterate the defect. The technical problems encountered are illustrated by the following case reports.

REPORT OF CASES

CASE 1. A 54 year old man was admitted to the clinic for evaluation of a tumor of the right chest which had been demonstrated by a recent radiologic survey (Fig. 3a and b). No other previous chest roentgenograms were available. He was asymptomatic.

Physical examination and laboratory studies were noncontributory. Roentgenography and fluoroscopy of the chest revealed a rounded soft tissue shadow at the right base, located medially and posteriorly. A preoperative diagnosis of neurofibroma was considered.

Right thoracotomy was carried out and this demonstrated a tumor, measuring 5 by 7 by 7 centimeters, in the posterolateral aspect of the right hemithorax. The lesion consisted of retroperitoneal fatty tissue which had herniated through the diaphragm by way of a diaphragmatic defect. This defect was somewhat triangular shaped with its base at the twelfth rib and measured 5.5 centimeters in its greatest diameter. There was a pleural covering but no peritoneal sac. The fatty tissue was excised, thus exposing the right kidney and adrenal below. The diaphragmatic defect was easily obliterated by means of a two layer closure (Fig. 3c) uniting the diaphragmatic margin to the chest wall. The postoperative course was uneventful.

CASE 2. A 57 year old white man was seen at the clinic with complaints of pain in the right upper abdominal quadrant of 6 to 7 weeks' duration. The pain was steady at the right costal margin and occasionally extended around the costal margin as far as the midaxillary line.

Physical examination was not unusual. Roentgenographic studies of the gallbladder and bowel were negative. Chest roentgenograms revealed an egg-sized mass in the right lower posterior mediastinum. The clinical opinion was that it was a diaphragmatic hernia or possibly a neurofibroma.

Right thoracotomy was carried out through the bed of the seventh rib, revealing a herniation of properitoneal fatty tissue through the posterolateral portion of the diaphragm. The diaphragmatic defect again was

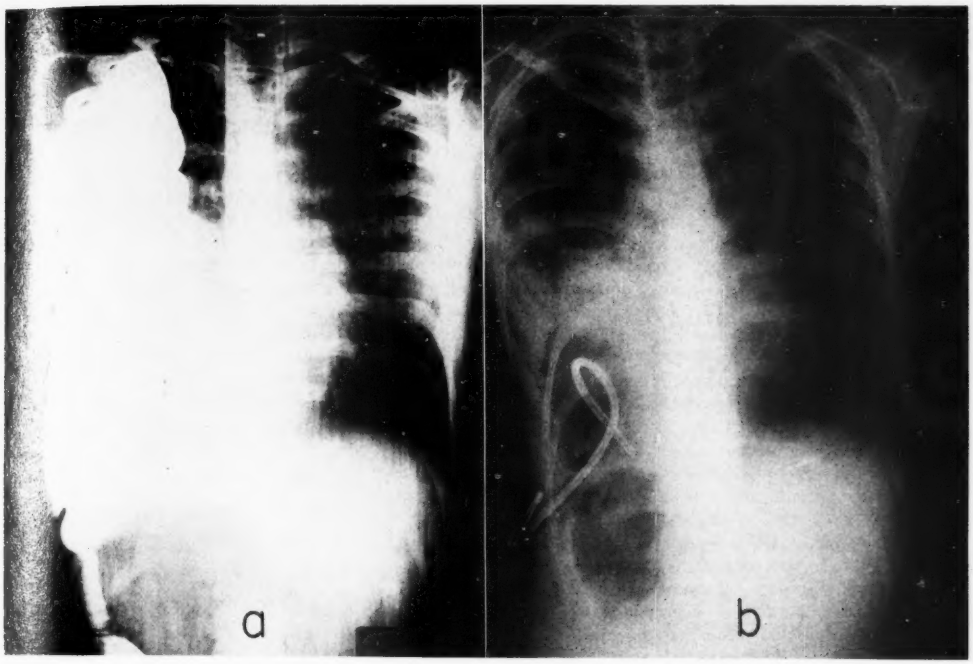

FIG. 4.

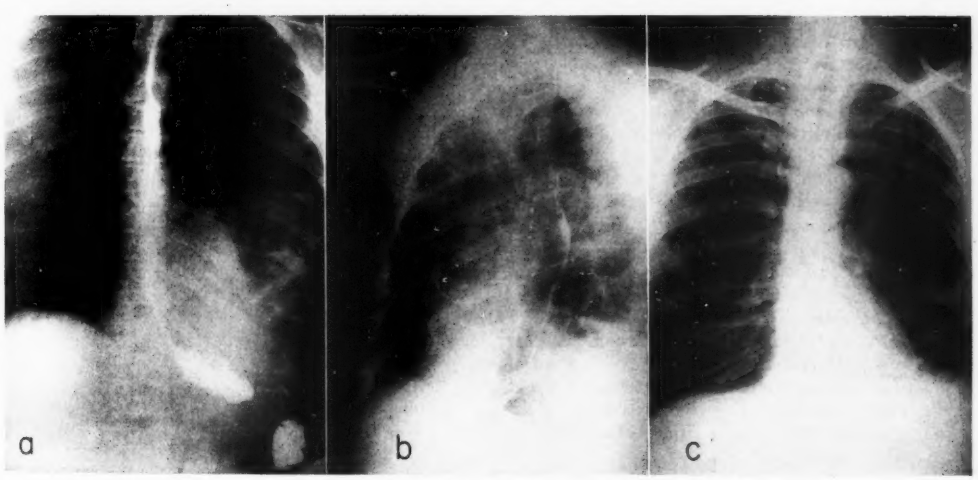

FIG. 5.

FIG. 4. a, A typical severe congenital hernia (many such patients die in infancy). b, Early postoperative film showing partial thoracoplasty and as yet incomplete expansion of the left lung.

FIG. 5. a and b, The confusing picture of a congenital diaphragmatic hernia in an adult. c, The postoperative result.

quite small and was easily obliterated with two rows of silk sutures. No component of peritoneum was involved in the hernia but the fatty tissue was overlaid in the chest with pleura. The postoperative course was uneventful and subsequent follow-up study revealed complete disappearance of the preoperative complaint of pain.

CASE 3. A 13 year old girl entered the clinic with a history of exertional dyspnea since the age of 3 years. Roentgenograms taken at the age of 7 were said to show partial collapse of the left lower lobe of the lung and elevation of the left diaphragm. She had been cyanotic on occasions and complained of intermittent pain in the left lower chest. This pain was aggravated by intake of food and somewhat relieved by lying flat. There was no history of trauma. Admission to the hospital was precipitated by an acute attack of cyanosis following a colonic irrigation.

Physical examination revealed flattening of the abdomen. There was some displacement of the trachea and heart to the right and diminution of breath sounds over the lower anterior aspect of the left chest. A reduction in the respiratory excursion of the left hemithorax was apparent. Gastrointestinal series revealed the small intestine and part of the large bowel to be in the left chest (Fig. 4a). The stomach was within the abdomen and was rotated 180 degrees.

Left thoracotomy was performed, revealing herniation of the entire small bowel distal to the ligament of Treitz and the colon proximal to the splenic flexure into the left chest. On opening the chest the appendix presented. There was no hernial sac and the diaphragmatic defect, permitting free communication between the peritoneal cavity and the thorax, was present posterolaterally. It measured 6 inches in its greatest diameter. There was compression atelectasis of the left lower lobe. Reduction of the herniated viscera was accomplished with some difficulty. Although the defect in the diaphragm was not extensive, approximation by direct suture could not be achieved. Phrenic nerve crush was carried out intrathoracically but it was necessary to perform, in addition, extraperiosteal resection of the left tenth, eleventh, and twelfth ribs to permit closure. The postoperative course was uneventful except for persistent wound infection, and re-expansion of the left lower lobe eventually was obtained.

CASE 4. A 65 year old white man entered the clinic with the complaint of chronic cough of 10 years' duration. Recently the cough had increased somewhat and for the past 12 months it had been productive of up to ½ cupful of mucoid sputum per day. He had noted exertional dyspnea in addition.

On physical examination bowel sounds were audible over the left chest in the lower anterior aspect. Roentgenograms of the chest and gastrointestinal tract revealed increased density of the left pulmonary base indistinguishable from the diaphragm (Fig. 5a and b). Bowel gas was seen within this increased density. The left diaphragm was limited in its excursion. Bronchoscopy and bronchograms revealed a collapsed left lower lobe.

Left thoracotomy was carried out through the bed of the seventh rib. The lower lobe of the left lung was completely collapsed. Herniation of the transverse colon, greater omentum, and spleen into the left chest was noted. This herniation had occurred by way of a large congenital posterolateral defect. No hernial sac, as such, was evident. Reduction of the intra-abdominal viscera was carried out and the diaphragmatic defect was closed. In order to achieve this closure satisfactorily a flap of fascia lata from the patient's thigh was employed.

Postoperatively there was good re-expansion of the entire left lung (Fig. 5c), and subsequent follow-up study revealed complete cessation of cough and other symptoms.

SUMMARY

The nature of congenital herniation through the posterolateral aspect of the diaphragm has been reviewed from a developmental standpoint. The infrequency of the occurrence of these hernias in adults is due to the high mortality which has always accompanied them in infancy and childhood. Symptoms produced are varied and numerous and are manifestations of the associated disturbances produced in the cardiorespiratory and gastrointestinal tracts. Four cases are presented. These cases illustrate the variations in symptoms and in degree of herniation.

The available surgical techniques are indicated and may include simple closure, autogenous fascial graft, and partial thoracoplasty.

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THE RESULTS OF SURGICAL PALLIATION IN 32 PATIENTS WITH TRANSPOSITION OF THE GREAT VESSELS

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TRANSPOSITION OF THE GREAT VESSELS, pulmonary artery and aorta, constitutes one of the gravest forms of congenital heart disease from the viewpoint of the natural mortality of the disease, and also from the viewpoint of its frequency. The formerly held concept, that this is a rare form of congenital heart disease, is disputed by several studies. A review of the literature reveals this defect to be present in 75 of 1,245 cases of congenital heart disease, or 6 per cent, while in this same series the tetralogy of Fallot was found in only 100 cases, or 8 per cent. Gardner and Keith have recorded this condition to be the second leading cause of death from congenital malformations of the heart. This misconception of the incidence of complete transposition may be explained by the fact that the defect may be easily, and probably is quite frequently, overlooked in the autopsy specimen. However, with the increased interest in the correct diagnosis of congenital heart disease which has been stimulated by the now rather widely used techniques of cardiac surgery, later series of autopsy analyses may show an even greater incidence of this malformation.

The gravity of the disease is well demonstrated in a series of 114 patients with complete transposition, which were analyzed by two of us (3), wherein it was noted that only 4 per cent of the series were alive at 7 years of age and 78 per cent of the group had expired during the first year of life. Blalock and Hanlon have noted, in their series of 123 collected cases, that the average length of life was 19 months and, further, if the few long term survivors, who probably had very large intracardiac shunts, are removed from the group, the average survival time is reduced to 5.5 months. On the other hand, stillbirth from this condition is extremely rare, as fetal circulation is not seriously impaired.

From the foregoing, it is obvious that, if any attempt at surgical intervention is to be carried out, it must take place at an early age and at the first signs of deterioration of the general condition of the patient (Figs. 1 and 2).

The surgical and physiologic considerations of this condition were discussed by two of us in 1953 (3), when it was proposed to convert this defect to that of a corrected transposition by anastomosing the pulmonary drainage of the right lung to the systemic auricle, the right auricle, and also to transplant the vena cavae to the pulmonary auricle, the left auricle (Figs. 3 and 4). Attempts have been made by Mustard and associates to correct this condition surgically by an interchange of the arterial pathways. It is believed that this procedure would be difficult, due to the origin of the coronary

TABLE I.—AGE AND DEGREE OF CYANOSIS AT OPERATION IN 32 PATIENTS

Age	*No. of patients*
0 to 1 mo.	5
1 to 6 mos.	12
6 to 12 mos.	5
1 to 2 yrs.	8
2 to 3 yrs.	1
3 to 4 yrs.	1
Degree of cyanosis	
Grade 1 (mild)	1
Grade 2	7
Grade 3	19
Grade 4 (severe)	5

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Dr. Murphy is a National Heart Trainee, U.S. Public Health Service.

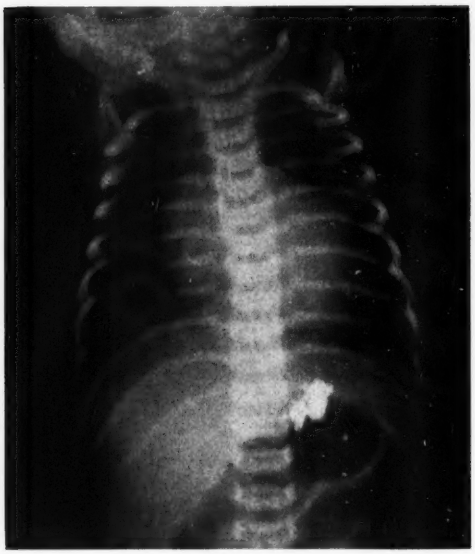

FIG. 1.

FIG. 2.

FIG. 1. The typical configuration of the heart with transposition of the great vessels.
FIG. 2. By angiocardiogram, the contrast medium is seen rapidly to enter the transposed aorta with only late, and a faint amount of, pulmonary visualization.

arteries from the base of the transposed aorta. If such a procedure were successful and the anastomosis were made distal to the coronary artery origins, it would result in a desaturated blood supply to the myocardium, in itself a very serious congenital defect.

The present report is an analysis of 32 attempts at surgical palliation of complete transposition, from March of 1952 until January 1955, at the University of Minnesota Hospitals.

TABLE II.—ASSOCIATED CONGENITAL DEFECTS

	No. of cases
Complete transposition	32
Interventricular septal defects	16
Patent ductus	11
Interauricular septal defects	10
Patent foramen ovale	10
Pulmonary stenosis	6
Overriding pulmonary artery	5
Overriding aorta	3
Hypoplastic aorta	2
Coarctation of the aorta	2
Hypoplastic pulmonary artery	1
Hypoplastic right ventricle	1
Levocardia	1
Bicuspid pulmonary valve	1

In this series there was a preponderance of male patients, 20 males and 12 females. The ages of the patients at the time of surgery as well as the degree of cyanosis are noted in Table I. Seventeen patients were operated upon during the first 6 months of life, and 22 of the 32 patients were operated upon during the first year of life. The factors in the consideration of a surgical procedure were: the degree of cyanosis, the stage and rate of enlargement of the heart, and the general physiologic growth and development that the child exhibited to the existence of a desaturated arterial blood supply.

Complete transposition of the great vessels cannot exist as a pure form of congenital heart disease for it is incompatible with life. Table II shows the associated congenital defects that make life possible. It has long been recognized that some sort of an intracardiac shunt is necessary to allow mixing of the otherwise isolated pulmonic and systemic circulatory systems. In Hanlon's review, he

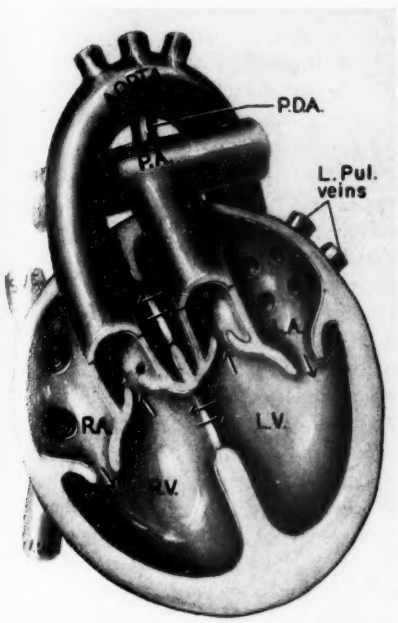

FIG. 3.

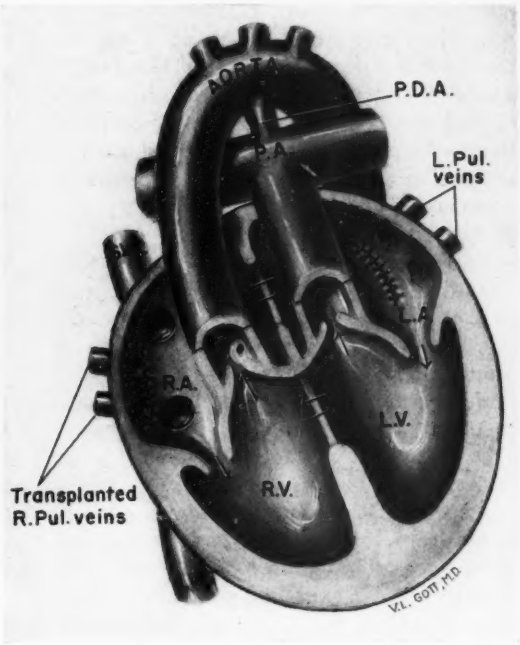

FIG. 4.

FIG. 3. Diagrammatic drawing of complete transposition of the great vessels with the commonly associated defects, patent ductus (*PDA*), interventricular septal defect, interauricular septal defect.
FIG. 4. Diagrammatic drawing of the surgical approach to palliation with transplantation of the right pulmonary veins to the systemic auricle.

has compared the type of shunt with the severity of the condition, and thus it can be shown that a transposition with a large interventricular septal defect has a better immediate outlook than with other types of shunt. A large interauricular defect is the second most favorable lesion. In all survivors in this series, in whom a surgical transplantation of the pulmonary drainage was done, an interventricular septal defect was diagnosed.

The following types of surgical procedure were attempted. The establishment of a venous shunt to the systemic (right) auricle by means of transplanting the right pulmonary vein was attempted in 10 patients, and a further attempt at correction by also transplanting either the superior or inferior vena cava to the pulmonic (left) auricle was attempted in 13 more patients. In 6 instances, in which there was a pulmonary artery stenosis present, either a Blalock or a modified Potts procedure was done to establish systemic arteriolization of the pulmonary vascular bed. In 1 patient, an attempt was made to repair a coarctation of the aorta and to ligate a patent ductus. In 2 instances, the patient expired before any procedure other than a thoracotomy could be done.

Of this series of 32 patients, 7 are alive and doing modestly well at the present time. Four of these are patients in whom the right

TABLE III.—POSTOPERATIVE DEATHS

Survival time	*No. of patients*
At surgery	5
0 to 24 hours	11
24 to 48 hours	2
2 to 10 days	1
10 to 30 days	1
1 to six months	4
6 to 12 months	1
Total	25

pulmonary veins were anastomosed to the systemic auricle, the right, and 3 are patients with pulmonary stenosis in which a systemic arteriolization of the pulmonary circuit was established. Four of the 7 patients who are living were operated upon at under 2 months of age, and all were under 4 years old at the time that surgery was performed.

Table III shows the survival period of the 25 patients who have expired. Five patients expired during the procedure and could not be resuscitated. Fourteen other patients expired during the immediate postoperative period. Six patients survived the surgical procedure but expired during the first postoperative year from heart failure and intercurrent respiratory infections.

At present, the 7 survivors have been followed from 15 to 36 months since surgery. They have all gained weight and approach a more normal existence. It is noteworthy that several of these children have rather suddenly learned to walk after recovering from the surgical procedure. The degree of cyanosis is definitely lessened although they still show mild cyanosis with exertion. The hemoglobin range is from 16 to 17.2 grams per 100 cubic centimeters of blood, and the limits of physical endurance while reduced are not markedly so.

DISCUSSION AND CONCLUSIONS

Even though surgery in this group of patients carries with it a high degree of mortality, the 7 surviving patients are representative of a salvage beyond that to be expected from the natural history of this crippling lesion. Four of these 7 survivors were critically ill at less than 2 months of age, when the surgery was performed.

A tentative classification for types of transposition is proposed: (1) transposition associated with ventricular septal defect; (2) transposition associated with ventricular septal defect and obstruction in pulmonary outflow; (3) transposition associated with intact ventricular system; and (4) levocardia with situs inversus.

The situation in a child with pulmonary stenosis and transposition of the great vessels is a more immediately favorable one, for here the heart has a large pulmonic pumping ventricle to systemic pumping ventricular shunt. There is an obligatory movement of oxygenated blood into the systemic circuit. Systemic arteriolization of the pulmonary vascular bed merely increases the volume of this former shunt, making the defect more compatible with life. Fifty per cent of these patients survived the operation and are doing well.

The technical difficulties of the operative venous transplant procedure are considerable. Moreover, these patients are small and the balance of circulation between systemic and pulmonary circuits is delicate. They can easily overload either with the development of fatal pulmonary edema or heart failure. Man is but awkwardly capable of achieving nature's precisely balanced flow rates.

However, the possibilities for eventually curing this anomaly are so great that there is a stimulus to continue and redouble one's efforts toward this end.

SUMMARY

The results of attempts at surgical palliation of 32 patients with complete transposition of the great vessels are presented with a listing of 7 survivors. The condition of a child with this malformation is desperate, and he should be considered for surgical treatment once the correct diagnosis has been made. Our hopes continue for complete correction of this lesion by use of newer cardiac by-pass techniques.

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EXPERIMENTAL AND CLINICAL USE OF BONE MILLED IN THE KITCHEN BLENDER

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THE SCIENTIFIC APPROACH to the study of bone healing and transplantation began with Ollier in 1858. He opened an avenue of research which has led to thousands of studies on the subject of bone and bone transplants. Although bone grafts are now widely used there are divergent opinions regarding much of the physiology of bone transplantation. But it is widely agreed that the impetus for incorporation of a graft is on the side of the host and the role of the transplant is largely passive.

The principles of bone graft technique encourage new bone formation on the part of the host and dictate the choice of the graft which will be most receptive to the new bone of the host. Proper preparation of graft site, close apposition, and firm fixation of transplant to host minimize injury to the budding capillaries of the host and encourage their growth into the transplant. A cancellous transplant is incorporated in the host and replaced more quickly than a cortical transplant. This may be due to the greater bulk in cortical bone but more likely occurs because cancellous bone offers more accessible surface area to the inroads of the host blood supply. Autogenous grafts are more desirable than homogenous grafts perhaps because they are less likely to be incompatible with the host. They will not cause a fibrous tissue response due to antigens or a difference in tissue specificity. The autogenous osteocytes which may survive transplantation are probably not a major factor in the superiority of autogenous bone because frozen autogenous bone in which no cells survive is also superior to frozen homogenous bone. When a graft is prepared with a bone saw we endeavor to produce as little heat as possible not to conserve osteocytes, but in order that the crystalline and organic components will not be altered making them less compatible with the host. Central portions of massive cortical grafts may remain nonviable years after the remainder of the graft has been functioning. The nonviable portion is usually that part least accessible to the host.

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In order to make bone grafts more accessible to the host many are now transplanted in the form of chips. The surgical production of these chips with or without a bone mill is tedious and the chips are large.

When we began to experiment with instruments which might be adaptable as a bone mill, the bone saw and the coffee grinder produced fine particles of bone but generated too much heat and burned the bone. A pencil sharpener mounted on a firm base produced good particles of bone but was tedious and wasteful of bone because the "stubs" had to be discarded. Finally kitchen instruments known variously as blenders, liquidizers, or osterizers were used.

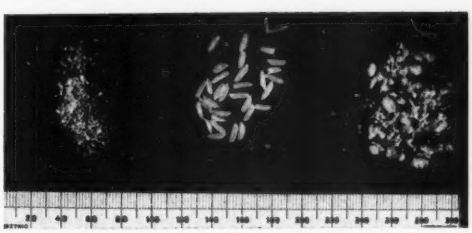


FIG. 1. a, Cortical bone which has been blenderized for 10 minutes. b, Grains of uncooked white rice. c, Cortical bone which has been blenderized for 5 minutes.

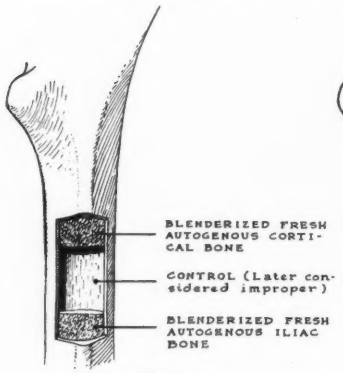

BLENDERIZED FRESH AUTOGENOUS CORTICAL BONE
CONTROL (Later considered improper)
BLENDERIZED FRESH AUTOGENOUS ILIAC BONE

FIG. 2.

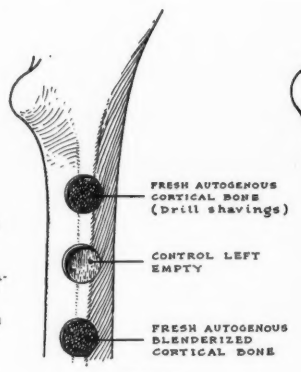


FIG. 3.

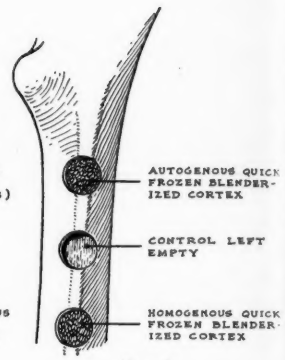


FIG. 4.

FIG. 2. Sketch of a long bone to show the method used in group 1.

FIG. 3. A sketch of a long bone with holes in the cortex made by a 3/8 inch crown drill as used in group 2.

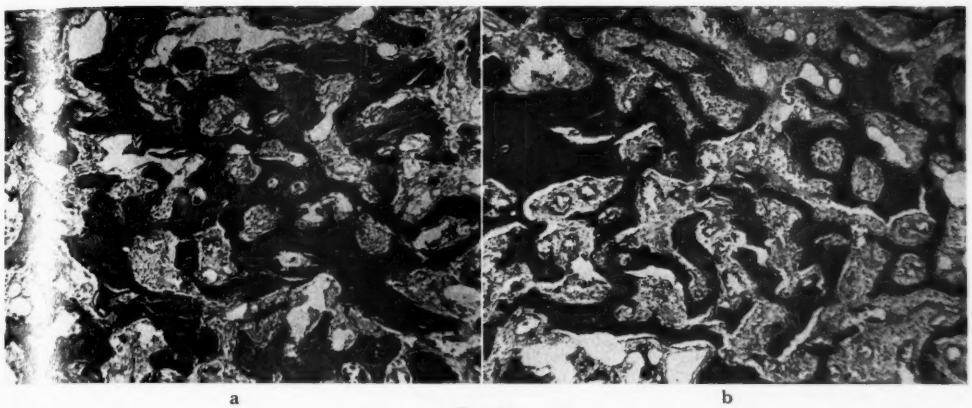
FIG. 4. Sketch showing defects created in the shaft of long bone by crown drills and filled, as described in group 3.

The instrument functions by the action of metal blades which spin rapidly in the bottom of the container when it is fitted on to the spinning axle of the motorized base. The electric motor cannot be sterilized but the container is autoclaved and its rubber or plastic top is cold sterilized. To determine whether the instrument could be used aseptically, sterile solution was placed in the apparatus which was then operated for 5 minutes, and cultures of the content were made. Ten consecutive trials showed no growth in the cultures.

The container and its top and the bone are handled by the surgeon. The motorized base is on a nonsterile table and is operated by the circulating nurse who pours the sterile solution. Two to 4 inches of chilled normal saline solution are placed in the sterile container. This serves the dual purpose of bringing the bone fragments into contact with the whirling blades and also of dissipating heat which is produced. The solution rarely surpasses 50 degrees centigrade, but if temperatures rise above this, more chilled saline may be added or the motor may be stopped until the solution cools. A piece of cortical bone, 8 by 2 by 1 centimeters, can be reduced to the consistency of coarse sand in about 5 minutes (Fig. 1). Many much smaller particles measuring only a few microns remain suspended in solution but these settle quickly so that the solution can be decanted and most of the bone can be recovered. The tissue bank at the National Naval Medical Center in Bethesda, Maryland, has used this method plus freeze drying so that all of the material is recovered.

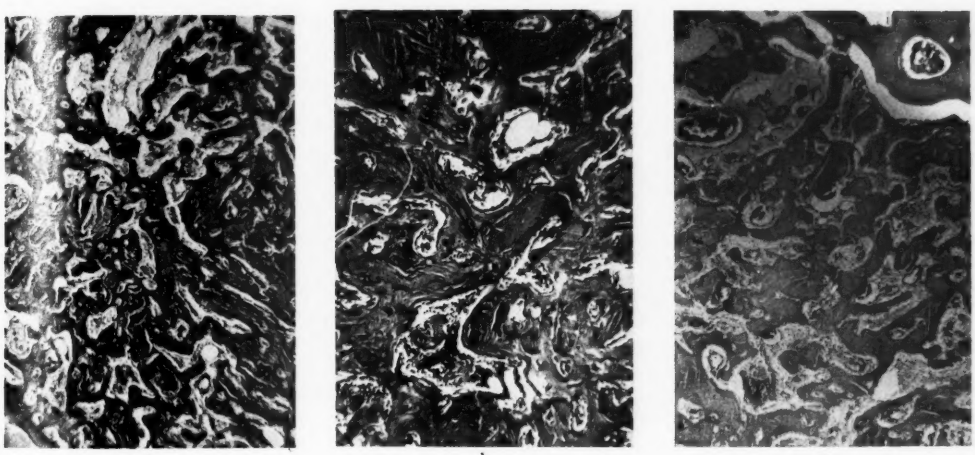
The ultimate size of the particles is determined by the size of the original piece of bone and the length of time in the blender. The blenderizing of bone washes the particles thoroughly and may destroy the osteocytes but probably does not chemically alter the organic or crystalline components of bone. Cancellous bone is reduced more slowly than cortical bone, perhaps because it offers less resistance to the whirling blades.

In 1950 Robinson, beginning with a sliver of cortical bone taken from a rib, was able to reduce it to hydroxyapatite crystals which have been established as the basic crystalline structure of bone (1). This had been determined by polarized light and x-ray diffraction studies but Robinson using the electron microscope was able to photograph these crystals (3). Subsequently in a series of brilliant submicroscopic studies he has shown



a b

FIG. 5.



a b c

FIG. 6.

FIG. 5. a, Photomicrograph of blenderized fresh autogenous cortical bone implanted in a femur and sectioned after 2 weeks. ×19. b, Photomicrograph of the section through an area of blenderized fresh autogenous iliac bone implanted 2 weeks before. ×19.

FIG. 6. a, Photomicrograph of blenderized fresh autogenous cortical bone implanted in a femur and sectioned after 21 days. ×19. b, Photomicrograph of the section through an area of blenderized fresh autogenous iliac bone implanted 21 days before. ×43. c, Cross section of normal femur. ×19.

that the C axis of the unit cell lies parallel to the long axis of the crystal and that the long dimension of the crystal lies parallel to the collagen fibers in normal bone structure. He has been able to establish that these crystals measure approximately 400 by 300 by 30 angstrom units (4). When we consider that 10,000 angstrom units equal 1 micron equals 1/1,000,000 of a meter it is not surprising that the surface area of 1 gram of bone reduced to basic crystals is probably over a hundred square meters.

His studies of the co-orientation of organic and inorganic components of bone have suggested to Robinson that orientation of the matrix of the bone would be destroyed by blenderizing if the process was carried to the ultramicroscopic level. If bone transplants

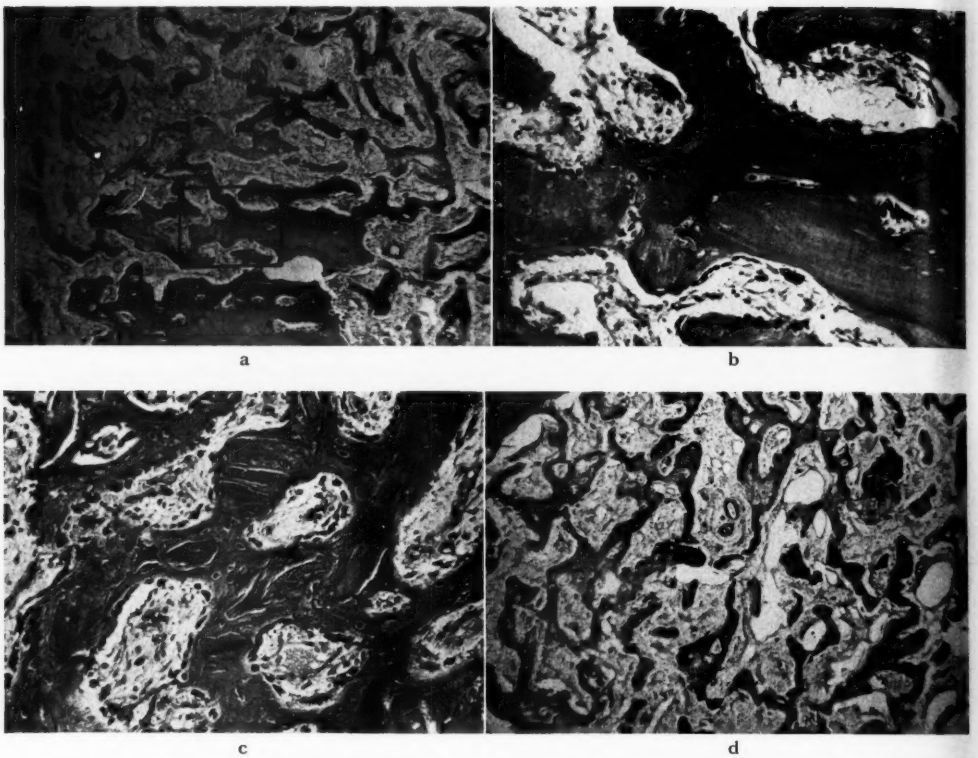


FIG. 7. a, Low power photomicrograph. ×21. b, High second power photomicrograph of section through a drill shaving specimen 3 weeks after implantation. ×120. c, High power photomicrograph of section through blenderized transplant after 3 weeks. ×60. d, Photomicrograph of control made at 3 weeks.

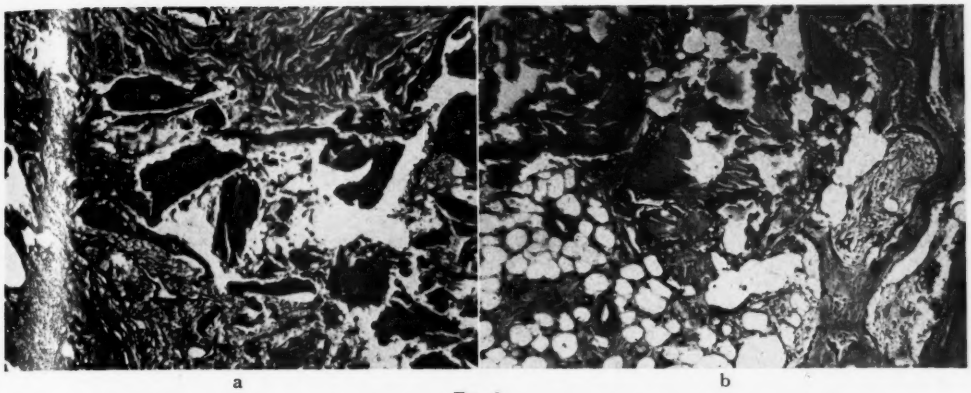
conduct the tissue of the host through their histologic patterns and bone is reduced to its basic crystalline structure these patterns would be destroyed and bone which had been blenderized to that extent might be of no value (2).

The blenderized particles which we have used have not been of uniform size but range from a few microns to 1 or 2 millimeters in diameter. Particles of this size provide a very much greater surface area to present to the host than a single piece of bone of equal bulk. Particles of this size do not violate microscopic tissue patterns but interrupt gross structure and by that means increase the accessibility of the microscopic patterns to the host.

EXPERIMENTS

We wished to determine by experimentation: (a) whether cortical and cancellous bone reduced to the same sized particles in the blender showed any difference when transplanted; (b) whether particles of blenderized fresh autogenous cortex would be incorporated in a host more or less quickly than somewhat larger particles of nonblenderized fresh autogenous cortex; and (c) the relative rate of incorporation of frozen homogenous and frozen autogenous blenderized particles.

The experimental animals in the entire study were mature dogs. The long bones utilized were only the humerus and the femur because these permit larger defects



a b

FIG. 8.

a b

FIG. 9.

FIG. 8. a, Photomicrograph of section through quick frozen homogenous blenderized cortex 2 weeks after implantation. ×48. b, Photomicrograph of section through autogenous quick frozen blenderized cortex 14 days after implantation. ×48.

FIG. 9. a, Photomicrograph of section through quick frozen homogenous blenderized cortex 28 days after implantation. ×48. b, Photomicrograph of cross section through quick frozen autogenous blenderized cortex 28 days after implantation. ×48.

than other long bones. Both femora and both humeri were used in all dogs. The defects were confined to the middle third of each bone. Occasionally fractures or infections occurred in which case the bone was excluded from the series. In all cases anesthesia was obtained with intravenous sodium pentobarbital. The preoperative preparation consisted of clipping the hair with surgical clippers and shaving with a razor. A thorough preparation of skin with soap and water, pHisoderm, alcohol, and aqueous zephiran.

METHODS

Group 1. Cortical defects 2 inches long and ¼ inch wide were created by means of the double-bladed bone saw. The material removed from the defect was denuded of cancellous bone, and the cortical portion was blenderized as described. Cancellous bone was taken from the ilium and blenderized. All cancellous bone was removed from the medullary canal and one end of the defect was packed with the blenderized cortical bone, the other end of the defect was packed

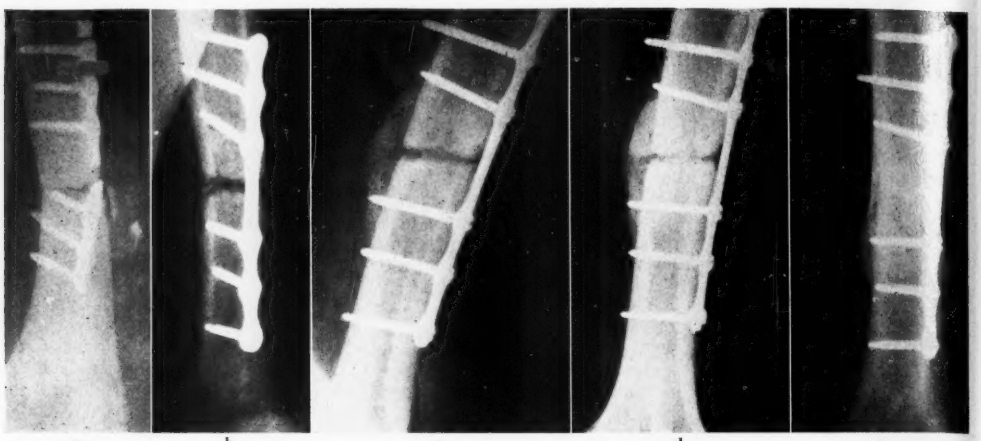


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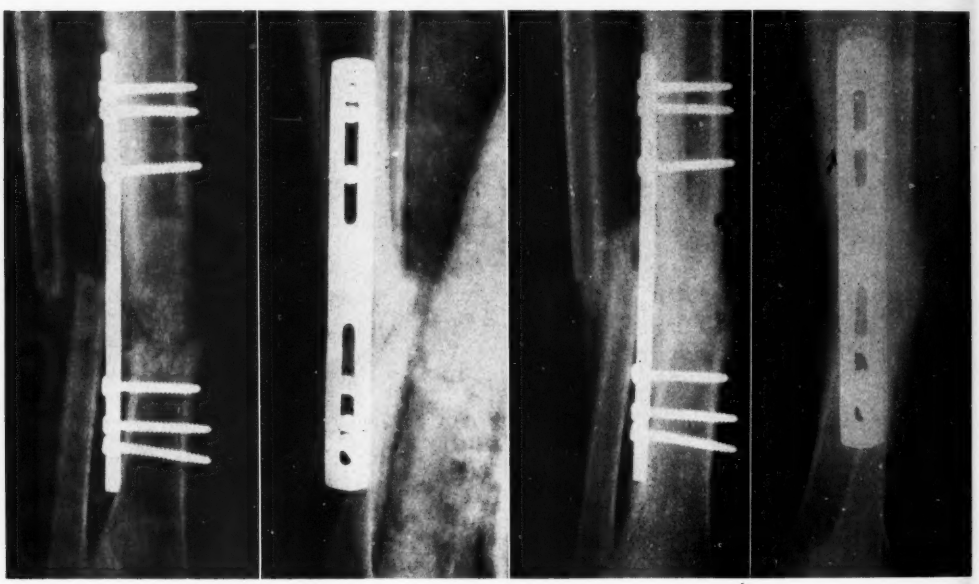
FIG. 10.



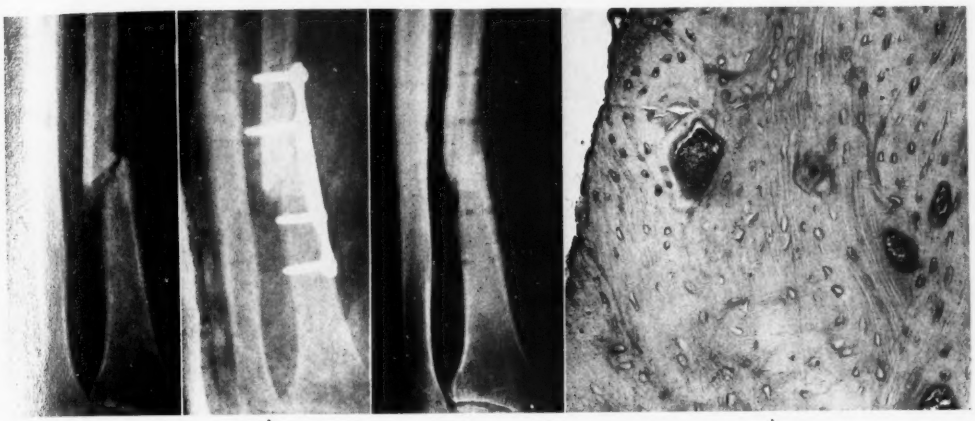
a b

FIG. 11.

FIG. 10. Case 1. a, Lane plate broke 3 weeks after open reduction for fractured humerus. b, Five weeks after second operation at which homogenous deep freeze blenderized cortex was added around the fracture site. c, Fifteen weeks after operation. d, Twenty-two weeks after operation. e, Sixty weeks after operation.

FIG. 11. Case 2. Delayed union of tibia due to improper immobilization. a, Postoperative anteroposterior and lateral films at which autogenous blenderized cortex has been used. b, Nine weeks postoperatively. Patient refused further treatment, but has been asymptomatic for 4 years.

with blenderized iliac bone (Fig. 2). The intervening space was originally considered to be control but was not satisfactory because the implants had been more completely surrounded by host than the controls and in subsequent experiments this was rectified. Most of the limbs were studied at 14 and 21 days.



a b c d

FIG. 12. Case 3. Nonunion of fractured radius. a, Six months following injury. b, Immediate postoperative film at which autogenous blenderized iliac bone was added. c, Twenty-six weeks postoperatively. c, Photomicrograph of section taken from graft site 24 weeks following implantation. ×23.

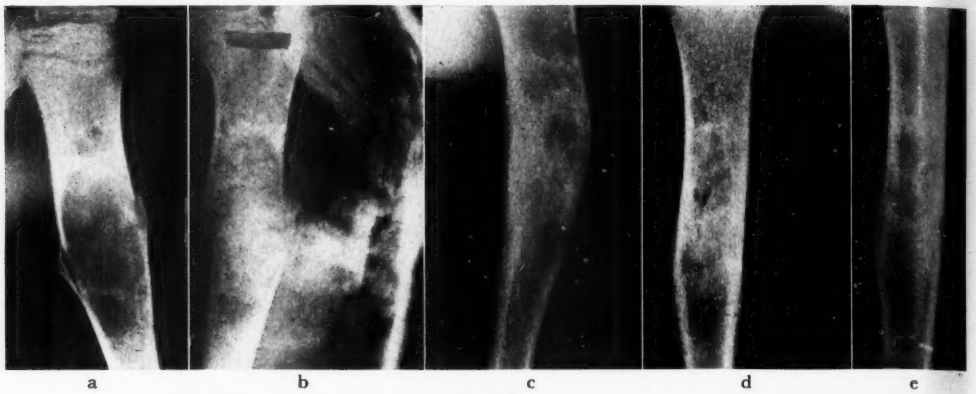
In the experiments which were terminated at the end of 14 days iliac and cortical bone, both of which had been blenderized, showed new growth of osteoid and young bone (Fig. 5). There was no discernible difference between the cortical and the iliac grafts. The bone pattern showed vestiges of the smaller particles but dead bone was present only in the large particles. These were surrounded by osteoid tissue and young bone. At 21 days the growth of new bone had progressed (Fig. 6). The appearance of particles of dead bone was less frequent and the mass of new bone was more dense than in the 14 day specimens. It was impossible to distinguish between the cortical and cancellous particles. Three dogs were used and 8 limbs were studied.

Group 2. Defects in the cortex were created by means of 3/8 inch crown drills turned slowly by hand (Fig. 3). Drill shavings were packed in one defect, blenderized autogenous cortical bone from the crown was packed into the second defect, and the third was left empty as a control. The drill shaving particles are larger than the blenderized particles but still are smaller than could be obtained with bone mills.

At 14 days both the drill shaving bone and the blenderized bone showed more dense formation of new bone than the control. There were small particles of dead bone surrounded by dense matrix of osteoid tissue in a pattern similar to but denser than a normal cross section. The pattern was heavier and much less delicate than that in the control. At 21 days there was ample evidence of dead bone among the larger drill shaving specimens (Fig. 7). In the blenderized specimen at this time there were barely perceptible small areas of dead bone completely surrounded by osteoid. Both of the specimens were far more dense than the control at this time and the pattern of new bone was far sturdier than the control. The small particles of bone obtained by blenderizing were more quickly incorporated in the host than the larger drill shavings. In this group 2 dogs were used and 7 limbs studied.

Group 3. Defects were created in the shaft of long bones by means of 1/4 inch and 3/8 inch crown drills turned by hand (Fig. 4). The bone obtained was quick frozen, blenderized, and placed in one drill hole. A second was left empty as a control and the third drill hole filled with similarly prepared homogenous cortical bone.

At 14 days there was a modest amount of new bone in the homogenous specimens (Fig. 8). There was fibrous matrix surrounding



a b c d e

FIG. 13.

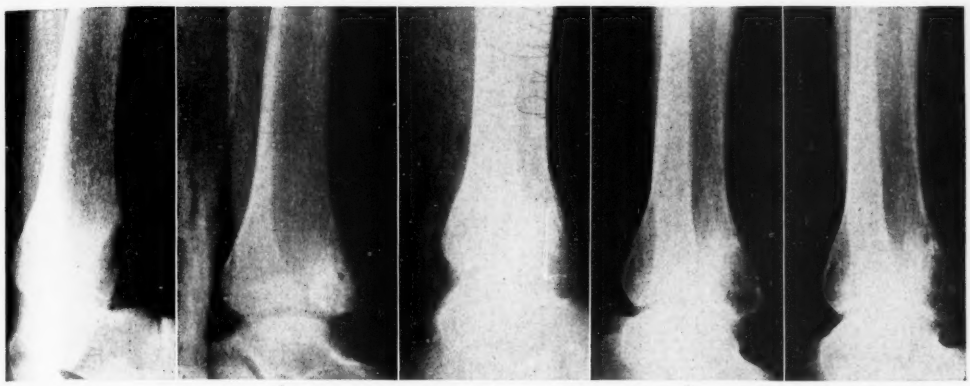
a b c d e

FIG. 14.

FIG. 13. Case 4. a, Fracture through unicameral cyst of humerus. b, One week later following implantation of blenderized homogenous deep freeze cortex. c, Twenty weeks postoperatively. d, One year postoperatively. e, Two and a half years postoperatively.

FIG. 14. Case 5. a, Fracture through unicameral cyst of humerus. b, Six months later fracture had healed but cyst had not. c, Immediate postoperative film following implantation of blenderized homogenous deep frozen cortical bone. d, Seven months postoperatively. e, Sixteen months postoperatively.

the dead particles of homogenous bone and osteoid tissue was minimal. In the autogenous specimens there were areas of dead bone which were surrounded by osteoid tissue. The autogenous specimens were more dense and there was less fibrous tissue than in the homogenous specimens. The impetus from the host was of good quality in both homogenous and autogenous specimens in that the response to the operative trauma was the same in both areas but the homogenous graft seemed to repel this growth (Fig. 9). At 28 days the homogenous specimens showed more fibrosis. There was an increase in the amount of osteoid tissue and more new bone was present but there was still dead bone in the homogenous specimens whereas the autogenous specimens no longer showed any evidence of dead bone. The homogenous specimen was slower than the controls in 3 limbs of one 12 year old dog. The autogenous transplants were also somewhat slower in



a b c d e

FIG. 15. Case 6. a, Giant cell tumor distal tibia. b, Immediate postoperative film following implantation of blenderized autogenous fresh cortical bone. No roentgen therapy. c, After 6 months much of the transplant appears to have absorbed. d, At 8 months there is new bone formation and the cyst is smaller. At this time immobilization was discontinued and the patient allowed normal activity. e, One year postoperatively. Patient is doing heavy labor and is asymptomatic though defect is still present.

this animal but were ahead of the controls. In this group 4 animals were used and 11 limbs studied.

THE RESULTS OF THE EXPERIMENTS

1. There was no discernible difference in the behavior of cancellous and cortical bone when both had been blenderized. When the bone was reduced to similar sized particles cancellous grafts were indistinguishable from cortical grafts. The smaller particles were quickly replaced and only an occasional larger particle could be seen to remain of either iliac or cortical transplants. This seemed to indicate that clinically we could employ either cortical or cancellous blenderized bone with the expectation of similar results.

2. Autogenous cortical drill shavings promoted the formation of osteoid tissue in new bone as well as the blenderized cortical autogenous specimens; however, the drill shavings were larger and were still identifiable in the graft site at 21 days when practically no blenderized bone could be recognized. We considered that clinically we might use the blenderized particles as well or better than handmade or milled chips.

3. Frozen homogenous bone was less acceptable to the host and showed more fibrous tissue reaction than frozen autogenous bone even though both had been blenderized. A constantly evident factor was that the impetus to new bone formation came entirely from the host. The impetus took the form of granulation tissue which was rapidly transformed to fibrous then osteoid tissue in the autogenous transplants; however, in homogenous transplants the reaction resembled a foreign body reaction in that there was more fibrosis and the osteoid and bone developed later. The increased surface area of blenderized homogenous bone might have multiplied any difference in tissue specificity.

CLINICAL MATERIAL

In view of the experimental results it was believed that bone milled in the blender warranted a clinical trial. The blenderized bone was first used as a supplement to open reduction in the treatment of fractures. It was placed around the fracture site but not between the living fragments. Later it was added to other bone grafts in the treatment of nonunion and occasionally as the only transplant with a metallic plate and screws:

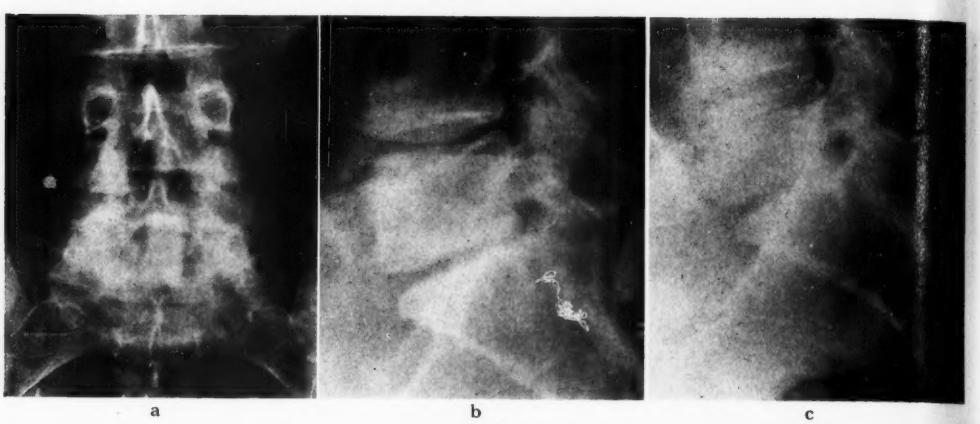


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FIG. 16.

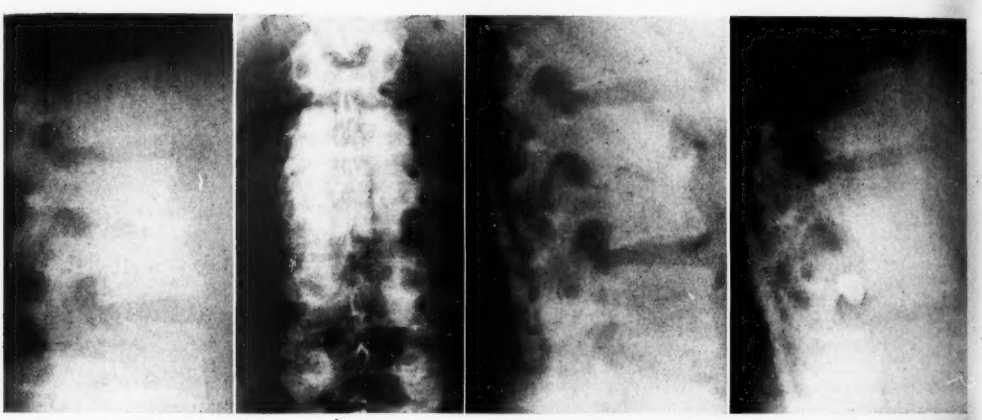

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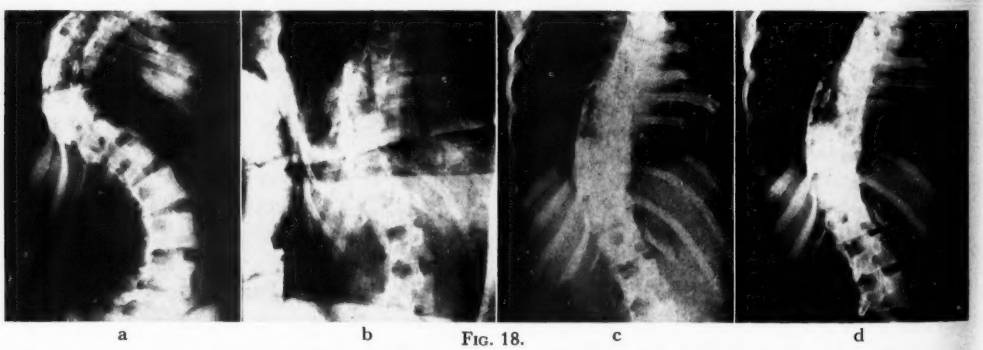
FIG. 17.



a b c d

FIG. 18.

FIG. 16. Case 7. Lumbosacral fusion following laminectomy for herniated disc. a, b, Anteroposterior and lateral films postoperatively showing cortical latch graft and blenderized particles. All bone was frozen homogenous cortical bone. c, Lateral film 8 months postoperatively. Patient has been asymptomatic for 2½ years.

FIG. 17. Case 8. Lumbar tuberculous spondylitis. a, Lateral preoperative film. b, c, Anteroposterior and lateral postoperative film showing latch graft and blenderized particles and overlying cortical grafts. All bone was frozen homogenous cortex. d, Lateral film 1 year later.

FIG. 18. Case 9. Idiopathic scoliosis. a, Preoperative film. b, Immediate postoperative film showing correction of the major curve. c, Seven months postoperatively. d, Sixteen months postoperatively. The only bone which was added was homogenous freeze dried blenderized cortical bone supplied by the U. S. Naval Medical School in Bethesda, Maryland.

FIG. 19.

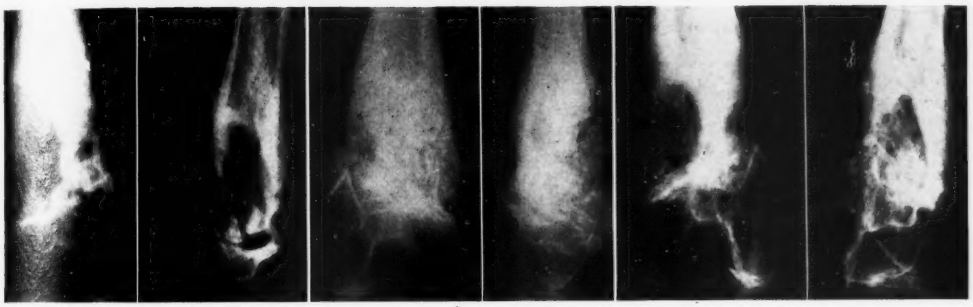


FIG. 20.

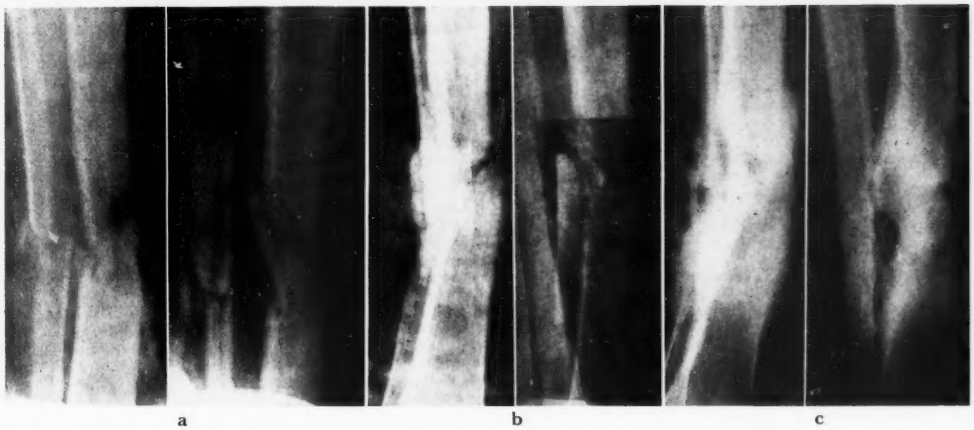


FIG. 21.

FIG. 19. Case 10. A tibial calcaneal fusion for painful astragalectomy. a, b, Anteroposterior and lateral postoperative films showing cortical graft and blenderized homogenous deep freeze cortical bone. c, d, Ten and 16 months postoperatively. Fusion is clinically solid and patient has been weight bearing without pain for 8 months.

FIG. 20. Case 11. a, Osteomyelitis. b, Postoperative following sequestrectomy and implantation of blenderized homogenous cortex. c, One year postoperatively.

FIG. 21. Case 13. Nonunion following compound fracture of the tibia. a, Preoperative. b, Anteroposterior and lateral postoperative films showing solid piece and blenderized particles of deep frozen homogenous cortical bone. c, Anteroposterior and lateral 8 months postoperatively.

TABLE I.—SUMMARY OF CASES

	Case	Age, years	Condition	Operation	Blenderized bone	Result
1	B.D.	64	Delayed treatment fractured humerus	Lane plate and blenderized bone	H, DF cortex	Satisfactory
2	L.B.	58	Delayed treatment fractured tibia	Eggers plate and blenderized bone	A cortex	Satisfactory
3	H.L.	26	Delayed treatment fractured radius	Lane plate and blenderized bone	A iliac	Satisfactory
4	J.L.	9	Unicameral cyst humerus	Blenderized bone	H, DF cortex	Satisfactory
5	H.B.	8	Unicameral cyst humerus	Blenderized bone	H, DF cortex	Satisfactory
6	L.R.	17	Giant cell tumor distal tibia	Blenderized bone	A cortex	Doubtful
7	E.E.	49	Herniated lumbar disc	Homogenous latch, blenderized bone	H, DF cortex	Satisfactory
8	N.C.	36	Lumbar spine tbc.	Homogenous latch, blenderized bone	H, DF cortex	Satisfactory
9	N.F.	15	Scoliosis	Hibbs fusion with blenderized bone	H, FD cortex	Satisfactory
10	M.V.	21	Painful astragalectomy	Tibiocalcaneal arthrodesis with blenderized bone	H, DF cortex	Satisfactory
11	E.C.	27	Osteomyelitis	Sequestrectomy, blenderized bone	H, DF cortex	Satisfactory
12	J.K.	31	Osteomyelitis	Sequestrectomy, blenderized bone	H, DF cortex	Failure
13	B.M.	18	Osteomyelitis, compound frac. tibia	Sequestrectomy, cortical graft, blenderized bone	H, DF cortex	Satisfactory
14	R.H.	19	Nonunion femur	Homogenous cortex, blenderized bone	H, DF cortex	Satisfactory
15	M.T.	55	Nonunion femur	Homogenous cortex, blenderized bone	H, DF cortex	Satisfactory
16	O.J.	37	Delayed union tibia	Homogenous cortex, blenderized bone	A cortex	Satisfactory
17	S.B.	61	Nonunion tibia	Homogenous cortex, blenderized bone	A cortex	Satisfactory
18	E.L.I.	58	Delayed union tibia	Homogenous cortex, blenderized bone	A cortex	Satisfactory
19	P.S.	11	Unicameral cyst calcaneus	Blenderized bone	H, DF cortex	Satisfactory
20	H.G.	12	Unicameral cyst calcaneus	Blenderized bone	H, ED cortex	Satisfactory
21	M.O.	23	Spondylolisthesis	Homogenous latch fusion, blenderized bone	H, DF cortex	Satisfactory
22	F.P.	29	Herniated lumbar disc	Homogenous cortex fusion, blenderized bone	A cortex	Satisfactory
23	H.P.	48	Spondylolisthesis	Homogenous cortex fusion, blenderized bone	H, DF cortex	Satisfactory
24	M.R.	14	Scoliosis	Hibbs fusion, blenderized bone	H, FD cortex	Satisfactory
25	D.K.	40	Lumbosacral instability	Homogenous cortex fusion, blenderized bone	H, FD cortex	Failure
26	S.K.	38	Lumbosacral instability	Homogenous cortex fusion, blenderized bone	H, ED cortex	Satisfactory
27	R.C.	23	Fractured femur	Intramedullary nail, blenderized bone	H, DF cortex	Inadeq. follow-up
28	B.M.	54	Fractured femur, nonunion	Homogenous cortex, blenderized bone	A cortex	Satisfactory
29	R.C.	56	Fractured tibia	Olperd reduction-plaster, blenderized bone	H, FD cortex	Satisfactory
30	A.R.	64	Fractured radius and ulna	Intramedullary pins, blenderized bone	H, FD cortex	Inadeq. follow-up
31	W.A.	24	Fractured femoral neck, nonunion	Osteotomy, homogenous cortex, blenderized bone	A cortex	Satisfactory
32	M.M.	25	Spondylolisthesis	Homogenous latch, blenderized bone	H, DF cortex	Inadeq. follow-up
33	P.J.	17	Sacroiliac tbc.	Arthrodesis, blenderized bone	H, DF cortex	Satisfactory
34	G.K.	36	Lumbosacral instability	Homogenous latch fusion, blenderized bone	H, DF cortex	Satisfactory
35	S.D.	34	Delayed union fractured tibia	Homogenous cortex, blenderized bone	H, DF cortex	Satisfactory
36	T.M.	7	Cong. pseudarthrosis tibia	Homogenous cortex, blenderized bone	A cortex	Satisfactory
37	K.H.G.	17	Fractured radius and ulna	Intramedullary pins, blenderized bone	H, FD cortex	Inadeq. follow-up
38	M.W.	48	Nonunion femoral neck	Autogenous cortex, blenderized bone	A cortex	Satisfactory
39	E.A.	77	Nonunion femoral neck	Jewett nail, osteotomy, blenderized bone	H, DF cortex	Satisfactory
40	W.W.	21	Nonunion femoral neck	Autogenous cortex, osteotomy, blenderized bone	A cortex	Satisfactory
41	O.R.	66	Femoral neck fracture	Osteotomy and cortical graft, blenderized bone	H, DF cortex	Satisfactory

H—homogenous. A—autogenous. DF—deep freeze. FD—freeze dried.

Case 1, fracture (Fig. 10); Case 2, delayed union (Fig. 11); Case 3, nonunion (Fig. 12).

Results, while difficult to evaluate regarding the rate of healing or the part played by the graft, seemed to warrant a more extensive use of blenderized bone. The next group of cases in which it was used included the filling of defects caused by benign tumors: Case 4, bone cyst (Fig. 13); Case 5, bone cyst (Fig. 14); and Case 6, giant cell tumor (Fig. 15).

As the blenderized bone was employed successfully in a wider variety of cases it was applied in cases requiring spine fusion for different indications. In some cases it was combined with a latch graft which provided some degree of immobilization. In other cases it was the only bone supplement to a Hibbs type of fusion. The cases include Pott's disease, scoliosis, spondylolisthesis, and lumbosacral instability: Case 7, herniated disc (Fig. 16); Case 8, Pott's disease (Fig. 17); and Case 9, scoliosis (Fig. 18).

With rare exception blenderized bone transplants were successful and warranted broader and more frequent use. Occasionally we have waived some of the principles of bone grafting to employ blenderized bone when there was little reason to hope that any graft might be successful. These cases have usually presented large defects, sometimes infected, which have been filled with blenderized homogenous bone. Some have been successful and others have been unsatisfactory: Case 10, tibiocalcaneal fusion (Fig. 19); Case 11, osteomyelitis (Fig. 20); and Case 12, compound fracture of the tibia with osteomyelitis (Fig. 21).

Our small series of 41 cases, Table I, does not warrant a statistical study although the follow-up is from 1 to 5 years. Twenty-nine cases were done with homogenous bone. Twenty-one of these employed freezer stored specimens and 8 were done with "freeze dried" bone supplied by the tissue bank of The National Naval Medical Center. Twelve transplants were autogenous. One of these was iliac and the remainder cortical bone. Four patients could not be followed. There were 3 failures. One of these occurred with the use of autogenous cortical bone in a giant cell tumor (Case 6). Clinically the lesion was diminished in size and the patient is asymptomatic. One occurred when a hundred cubic centimeters of freezer stored homogenous bone was blenderized to fill an osteomyelitic cavity (Case 12). Subsequently the bone was removed. The third failure occurred when freeze dried homogenous bone was used in a lumbosacral fusion (Case 25). The wound did not heal and the graft was removed though there was no clinical evidence of infection.

CONCLUSION

We believe that bone transplants should be in the form of small particles whenever stability is not a factor. The kitchen blender is a simple and efficient instrument for making bone into small particles.

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EVALUATION OF MITRAL AND AORTIC VALVULAR DISEASE BY LEFT HEART CATHETERIZATION

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WITH THE INTRODUCTION of right heart catheterization it soon became evident that defects and abnormalities of the right auricle and ventricle and their associated septa could be more accurately diagnosed. However, except for an estimation of left auricular pressure from the "pulmonary capillary pressure," little information could be obtained concerning lesions of the mitral and aortic valves. To facilitate study of alterations of these valves, Bjork performed left auricular catheterization with the introduction of a needle paravertebrally in the ninth interspace, passing it into the left auricle, as described by Bjork and associates and by Kent and associates. The published reports concerning this procedure have shown some correlation between pressure curves obtained and the valvular lesions present, but have not established adequate criteria for the differential diagnosis of mitral and aortic valvular disease. The present report is concerned with our experience in 25 patients who have been evaluated for cardiac surgery by means of left heart catheterization. We have found the procedure to be safe and simple, giving accurate information as to the presence and degree of mitral stenosis, mitral regurgitation, and aortic stenosis. The value of this information in equivocal candidates for operation is obvious. The method of performing left auricular and ventricular catheterization and a correlation of the pressure curves we have obtained, with the valvular changes found at operation, are the basis of this report.

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METHODS

Following minimal sedation with nembutal or demerol, the patient is fluoroscoped so that a metal marker is placed over the position of the left auricle, approximately 5 centimeters to the left of the midline in the eighth or ninth interspace (Fig. 1). This location is then marked on the skin with ink. The patient lies on his right side and is draped after antiseptic skin preparation. A No. 17 gauge, 15 centimeter needle is then introduced through a procaine wheal at the previously determined location. The point of the needle is directed anteromedially at an angle of 20 degrees with the vertebral column. After the tip has entered the subcutaneous tissue, the obturator is removed and the needle is advanced, infiltrating 1 per cent procaine in its path. Should the esophagus be encountered the patient will often complain of burning throat pain. When the auricle is encountered, one will feel a distinct thrust against the needle with each auricular contraction. After the auricle is entered, direct auricular pressure readings can be obtained. These have been measured with a Sanborn electromanometer and were recorded with a simultaneous electrocardiogram on a Sanborn polyviso direct writing recorder. A No. 442T sterile polythene catheter is introduced into the auricle through the needle and thence into the ventricle. Attention to the needle bevel, curve of the catheter, and its manipulation has made passage of the catheter into the ventricle possible in all but 4 cases done early in this series. Pressures must be recorded continuously while the catheter is being advanced as it may stay in the ventricle for only a few beats if marked mitral regurgita-

tion is present. It is important to withdraw the catheter and needle as a unit should resistance be encountered upon withdrawing the catheter separately.

RESULTS

A total of 25 patients have been catheterized by this method. Of these, 4 had tight mitral stenosis, 8 had moderate stenosis and regurgitation, and 2 patients had little mitral valve involvement in spite of severe subjective symptoms. Nine patients had marked regurgitation. There were 4 patients with aortic stenosis, 2 of whom had associated mitral valve disease. All lesions have been verified at operation or autopsy except those of 7 patients with mitral regurgitation or minimal disease who were not operated upon and of 2 with pure aortic stenosis, 1 of whom is awaiting surgery at the present time. This latter patient is interesting in that he had been refused surgery 2 years previously because of his marked "mitral regurgitation." In our experience there has been no significant disparity between the valvular lesion as predicted by the pressure tracings and that found at operation.

We have experienced no mortality nor significant morbidity in this series of cases. At operation, several days following the left auricular puncture, there has frequently been a small amount of serosanguineous fluid in the pericardium. There have been no instances of clinically recognizable pneumothorax, hemothorax, or cardiac tamponade. One patient retained a small segment of polythene catheter in her left ventricle when it sheared off on the tip of the needle. It could be palpated at valvotomy but not extracted. There have been no recognizable deleterious effects produced by the presence of the polythene segment in the left ventricle.

During catheterization, the inadvertent entry of the needle into the left ventricle in 1 case and into the pulmonary artery in 2 other cases was not associated with any sequelae.

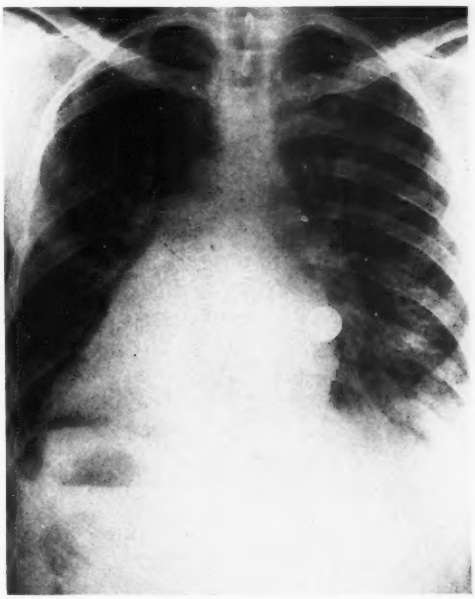

FIG. 1. Roentgenogram showing the position of the metal marker which had been placed over the left auricle in the right ninth interspace in a patient with mitral regurgitation. He had been explored 3 years before.

DISCUSSION

For the purpose of orientation it is necessary to describe the normal auricular pressure curve and its relationship to pressure changes in the left ventricle in the manner of Best and Taylor. In Figure 2 the auricular curve, which is not too different from the normal, can be seen to be made up of three positive waves. The first, or *A* wave, is caused by auricular systole. The second, or *C* wave, is caused by the rising pressure in the ventricle with the onset of ventricular systole. This pressure is transmitted to the auricle through the mitral valve. The negative wave following the *C* wave, the so-called "descent of the base," is produced by two factors: (1) the negative intra-auricular pressure caused by the ejection of blood from the ventricle; and (2) by the drawing caudad of the atrioventricular septum as the ventricle contracts. The third positive wave, the *V* wave, is caused by the inflow of blood

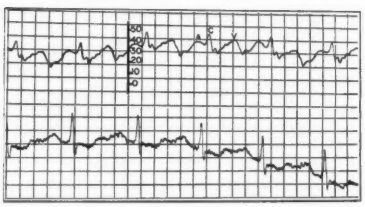

FIG. 2.

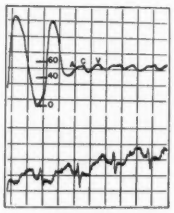

FIG. 3.

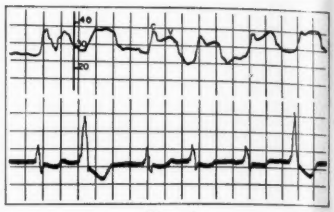

FIG. 4.

FIG. 2. This illustrates the three left auricular pressure waves characteristic of a patient with a tight mitral stenosis and a sinus rhythm. Note the descent of the base as manifested by a drop in pressure between the *C* and *V* waves.

FIG. 3. This tracing is on the same patient as in Figure 2. A record was taken as the catheter tip was withdrawn from the left ventricle into the left auricle. Note the high auricular as compared with the ventricular diastolic pressure.

FIG. 4. This record illustrates the left auricular pressure curve in a patient with a combined tight mitral and aortic stenosis. The essential diagnostic features of the tracing were not altered by the presence of the aortic stenosis. The *A* wave is absent in the presence of auricular fibrillation.

into the auricle from the pulmonary veins. The decline in pressure following the *V* waves occurs when the mitral valve opens. During ventricular diastole, the end diastolic pressures in the auricle and ventricle are equal.

In a patient with mitral stenosis of a severe degree and little or no regurgitation, the auricular pressure curve is very similar in shape to the normal as seen in Figure 2. It is to be noted that the descent of the base is maintained. In addition, the rise of the *V* wave is minimal and does not rise above the *C* wave. The diagnostic feature of the auricular curve in mitral stenosis is the high end diastolic pressure gradient between the auricle and ventricle (Fig. 3). This gradient can be measured by continuous pressure recording during left auricular catheterization as the catheter is withdrawn from the ventricle to the auricle. The pressure gradient depends upon the severity of the stenosis and the rate of flow of blood through the mitral valve during diastole. In the absence of measurements of cardiac flow, one cannot calculate the degree of mitral stenosis; however, one can make the general statement that the greater the pressure gradient across the mitral valve during diastole, the greater the stenosis. In those patients catheterized and found to have a tight mitral stenosis, this pressure gradient was found to be in excess of 15 millimeters of mercury.

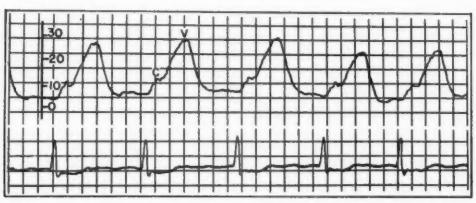

FIG. 5. This patient had a "pure" mitral insufficiency. The left auricular curve illustrates the low end diastolic pressure of 7 millimeters of mercury and the high *V* wave with an absence of the normal fall in pressure resulting from the descent of the base of the heart.

It is important to be certain that an associated aortic valvular lesion does not alter the diagnostic features of the left auricular pressure curve in mitral stenosis. In 4 patients with combined aortic and mitral lesions, the auricular tracing showed the characteristic features of the mitral lesion present. Figure 4 illustrates this in a patient with mitral and aortic stenosis who subsequently had a successful combined mitral and aortic valvulotomy. It is to be noted that the *V* wave does not rise above the *C* wave and that there is a high auricular and diastolic pressure of approximately 30 millimeters of mercury. The auriculoventricular pressure gradient was 22 millimeters of mercury.

The pressure curve obtained in patients with mitral insufficiency is also character-

istic. As seen in Figure 5, the diagnostic features are (1) the absence of the descent of the base following the *C* wave, and (2) the high *V* wave which begins somewhat earlier than normal, and rises higher than the *C* wave. In patients with marked and predominant mitral insufficiency, the difference in pressure between the *C* and *V* waves has been above 12 millimeters of mercury. Since in general the greater the amount of mitral regurgitation the less the stenosis, in cases of "pure" insufficiency one would not expect a high diastolic pressure gradient between the auricle and ventricle. This was verified in these patients with gradients ranging from 0 to 7 millimeters of mercury.

Between these two extremes are those patients with variable amounts of scarring of the mitral valve and all combinations of mitral stenosis and insufficiency. It is in this last group of patients that a purely clinical evaluation is most difficult and in whom left auricular puncture is of greatest value. By estimating separately the severity of the stenosis and of the insufficiency with the previously described criteria, we have been able to predict with a reasonable degree of accuracy the amount and type of valve deformity found at operation. Figure 6 illustrates a pressure tracing in a patient with some mitral regurgitation but predominant stenosis. The end diastolic pressure gradient of 11 millimeters of mercury between the

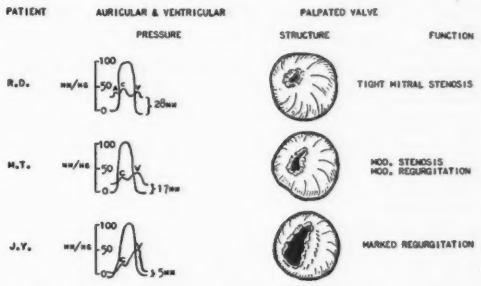

FIG. 7. Correlation of pressures and pathology. Auricular and ventricular pressure curves are superimposed to illustrate graphically the *V* wave changes and end diastolic pressure gradients as correlated with findings at operation.

auricle and ventricle indicates moderate stenosis. In addition there is little or no "descent of the base" which, along with a slightly elevated *V* wave arising early in ventricular systole, indicates some insufficiency.

Figure 7 graphically illustrates the superimposed pressure curves of the auricle and ventricle as correlated with the findings at operation in 3 patients with mitral valvular disease. One can see the relationship between the severity of the stenosis and the height of the end diastolic pressure gradient. It is also to be noted that the height of the *V* wave varies inversely with this gradient.

Of even greater difficulty than the differentiation of mitral valvular deformity is the evaluation of the degree of aortic stenosis

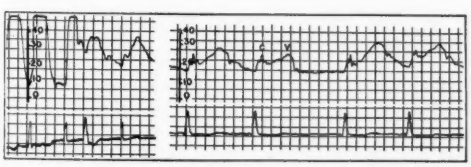

FIG. 6. This patient had some mitral regurgitation with a predominant mitral stenosis. The presence of the mitral stenosis can be predicted from the gradient between the end diastolic pressures of auricle and ventricle. The mitral insufficiency produces the high *V* wave and the small drop in pressure between the *C* and *V* waves. Also note in the second graph the high *V* wave in the third complex resulting from the longer diastolic pause.

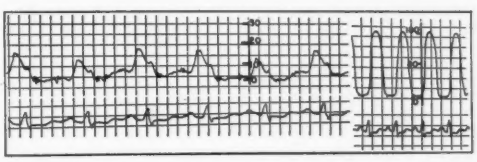

FIG. 8. These tracings were taken on a patient with aortic stenosis and minimal mitral stenosis. The auricular curve is fairly normal except for the giant *A* wave. This *A* wave is similar to that seen in right auricular pressure curves of patients with pulmonic stenosis. The peripheral systolic pressure was 100 millimeters of mercury and the ventricular systolic pressure was 180 millimeters of mercury, demonstrating a gradient of 80 millimeters of mercury across the aortic valve.

which may be present. In the presence of aortic lesions, left heart catheterization is desirable if surgical treatment is contemplated. As there is little correlation between the intensity of the murmur and the degree of aortic stenosis, it is very important to know the systolic pressure gradient across the aortic valve as an indication of the amount of obstruction its stenosis has produced. Figure 8 demonstrates the left ventricular pressure curve in a patient with aortic stenosis. The systolic pressure in the left ventricle was approximately 180 millimeters of mercury. This means that a pressure difference of 80 millimeters of mercury was needed to force the blood through the aortic valve during systole. The authors have not had sufficient experience with aortic stenosis to set an arbitrary pressure gradient that could be used as an indication for operation. The 4 patients with aortic stenosis who were studied had gradients greater than 70 millimeters of mercury. It is also to be noted that in Figure 8 the auricular curve is normal and the auricular and ventricular end diastolic pressures are identical, indicating the presence of a normal mitral valve.

The giant *A* waves recorded here have been seen in 2 cases and are similar to the large right auricular *A* waves seen in pulmonic stenosis.

SUMMARY

There is a need for estimating objectively the physiologic effects of valvular lesions on the left side of the heart.

Our experience in 25 cases justifies the application of left heart catheterization to this evaluation. It is a safe and simple method of evaluating mitral and aortic valvular disease.

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EXPERIENCES IN THE TREATMENT OF SURGICAL PATIENTS HAVING ANURIA AND UREMIA

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ANURIA AND UREMIA are complications that may occur in every phase of surgery and may present problems to every surgeon or surgical specialist. Irrespective of the basic cause of the complication, treatment is guided by 4 cardinal principles: (1) maintenance of body fluid; (2) correct control of electrolytes; (3) suppression of protein breakdown (forced high caloric, low protein diet); (4) avoidance of infection. If these measures appear to fail, or carry too great a risk, then a fifth principle is followed: (5) elimination of retention products by means of (a) artificial kidney, (b) peritoneal lavage, or (c) intestinal lavage.

The application of these principles in the actual management of surgical patients having anuria and uremia will be demonstrated in case reports. For each specific case, 1 or 2 techniques (of the many that are used for that case) will be commented upon. Thus, since each case offers examples of different techniques, together they comprise a review of present-day management of uremia.

Case reports are grouped under the following headings: (1) surgery of the blood vessels and the heart, (2) general abdominal surgery, (3) gynecologic surgery and obstetrics, (4) genitourinary surgery, (5) blood transfusion. A summary of present-day management of uremia in surgical cases and unit-conversion tables are given at the end.

ANURIA AND UREMIA AFTER SURGERY OF THE BLOOD VESSELS AND THE HEART

Anuria After Thrombectomy, Embolectomy, or Grafts of the Lower Aorta and Iliac Arteries

CASE REPORT. A 44 year old man had a thrombus removed from the right iliac artery. After the operation, the left leg proved to be cold. An embolus was suspected and was removed; however, on the following morning, the circulation in that leg had not returned. The urinary output decreased to 350 cubic centimeters per day.

As the patient was too sick to undergo immediate amputation, the leg was packed in ice to reduce absorption of toxic materials from the necrotic tissues, and he was given 30 grams of carboxylic resin per day to absorb potassium in the intestinal tract. As he was unable to retain food by mouth, he was fed through a plastic cardiac catheter by a continuous drip of hypertonic invert sugar (travert). His clinical condition slowly deteriorated; the blood urea rose to 285 milligrams per hundred milliliters on the ninth day after operation. He was treated with the rotating type of artificial kidney; blood urea was reduced to 140 milligrams per hundred milliliters and he seemed improved. The urinary output reached 1 liter on the fourteenth day, but on the seventeenth day (Fig. 1), thrombosis of the right femoral artery occurred and he died.

This was a case of crush syndrome in which urinary flow in the patient had been restored to 1 liter, so that the prognosis seemed favorable, but progression of the underlying disease prevented recovery. The moderate hypertension found in this patient is a common finding in acute renal failure of any kind. Severe rises of blood pressure are observed in renal failure consequent to renal arterial thrombosis, such as may occur with atherosclerotic disease. Prompt renal endarteriectomy or extirpation of the ischemic kidney may save such a patient's life.

The artificial kidney served a dual purpose (5, 9): first, it removed urea, creatinine, uric acid, and other metabolites such as sulfates and phosphates from the blood stream; second, it corrected abnormalities of the electrolyte composition of the blood plasma. By selecting an appropriate composition of the dialyzing fluid, the patient's electrolytes can be changed at will.

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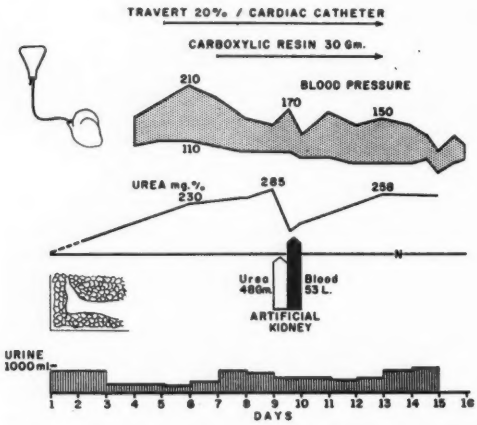

FIG. 1. Course of anuric patient with ischemic necrosis of 1 leg, followed by renal tubular necrosis. Hypertonic solutions of glucose or fructose were given by cardiac catheter.

The cardiac catheter was used as a last resort because the patient was unable to take food by mouth or gastric tube. It enables administration of glucose or fructose solutions as strong as 40 per cent, thus providing for 1,600 calories in the small volume of 1 liter (2). The tip of the cardiac catheter should lie in the superior vena cava or in the right auricle to insure that the hypertonic solution becomes mixed with the total body blood flow. About 100 milligrams of heparin per 24 hours are mixed with the infusion, to discourage formation of thrombi on the outside of the catheter, while its position prevents thrombosis or damage to the vein at its tip.

Anuria Following Mitral Commissurotomy

This case history offers an example of a type of patient best treated with the artificial kidney.

CASE REPORT. A 39 year old woman had had mitral stenosis for 16 years, with auricular fibrillation and increasing signs of cardiac decompensation for 11 years, and hemoptysis during the preceding 3 months.

A mitral commissurotomy was performed and, 8 hours later, it was first noticed that both legs were pulseless. After 3 injections each of 20 milliliters of 70 per cent urokon into the aorta, a large embolus was demonstrated by x-ray examination in the lower part of the aorta. It was surgically removed. Except for pain and hardening of the crural muscles during the first days after the second operation, the legs caused no further trouble and recovered completely; but, virtual anuria had set in and persisted for 18 days. On the sixth postoperative day she had ominous rales in the lungs and the abdomen was distended so that she was treated with the rotating type of artificial kidney. The following day she seemed improved; her appetite was good and she ate increased amounts of a high caloric low protein diet fortified with one of the commercial fat emulsions (6) (ediol or lipomul oral), 20 or 30 milliliters 3 times per day.

On the tenth day, as the level of the blood urea gradually climbed up again, she became acutely paranoid. This subsided when she was given a second treatment with the artificial kidney on the eleventh day. The urinary output slowly rose to 125 milliliters per 24 hours, but her general condition again deteriorated, and she developed convulsions and had cardiac arrest for 3 minutes on the fourteenth day. Dialysis became necessary again and on the fifteenth day she was treated with intermittent peritoneal lavage (11). The first day's lavage was quite successful: an inflow of 12 liters and an outflow of almost 13 liters resulted in a desirable loss of nearly 1 liter of fluid for the patient. The following day, however, because of leaking of peritoneal fluid into the subcutaneous space, the patient retained about 1,850 milliliters of fluid—a dangerous occurrence in a patient with oliguria. Fortunately, on the eighteenth day, diuresis increased to 450 cubic centimeters per 24 hours (Fig. 2), and on the twentieth day to more than a liter.

Her mental state gradually cleared, although she remained mildly suspicious for about 14 days, and she retained a slow, slurred speech for several weeks as evidence of organic brain damage. She had some discomfort in the shoulders and knees, which improved after 4 months. (Rheumatoid symptoms occur often after recovery from acute renal failure.) At that time she had recovered completely and felt better than at any time since she had developed the mitral stenosis.

The relation of urokon to the later development of anuria is problematic; there is increasing evidence that contrast media in high doses may be nephrotoxic.

This patient surely would not have recovered without the aid of dialysis. Peritoneal lavage was performed on the fifteenth day because it promised to be rather simple. The abdomen was soft and it was hoped that this procedure might tide her over the few days that probably separated her from the onset of diuresis. Although peritoneal lavage has been very useful in a great num-

ber of our patients, the unpredictability of its outcome is demonstrated in this case. It was not possible to recover all of the fluid instilled in the peritoneal cavity.

The toxic psychosis in this patient, as in others with uremia, varied in intensity with the level of retention products. It apparently is part of a generalized brain damage, which accounted also for convulsions on the fourteenth day. The latter were associated also with moderate hypertension (168/76).

Uremic convulsions usually are seen when blood pressure is higher than in this patient. The convulsions were suppressed with sodium amytal injected slowly intravenously. In this case, 120 milligrams were sufficient to stop the seizures. Barbiturates still are given in every case. When the convulsions are associated with crises of hypertension, reduction of the blood pressure is helpful: ansolysen, ½ to 2 milligrams intramuscularly as a single dose to be repeated, under control of blood pressure, every 1 or 2 hours. The room should be quiet and dark.

Her meals were carefully planned so that she was able to take the high caloric, low protein diet (7, 8) (Fig. 2). Such a "Borst diet" is important both to general well-being, and to reduce the protein breakdown. She had a slow rise in blood urea, notwithstanding her very extensive operations; without high caloric diet it would probably have been more.

The fluid balance was particularly difficult to maintain. She was in cardiac failure with pulmonary congestion from the beginning and was almost continuously in an oxygen tent, even during treatment with the artificial kidney.

She was overhydrated in spite of a low fluid intake, presumably by water of oxidation from the ingested calories. One should not give more than 700 milliliters of fluid per day to an anuric patient unless fluid is demonstrably lost with vomitus, drainage, or diarrhea. Oral sodium sulfate, 50 grams in 1 glass of lukewarm water was given to this patient on 1 occasion to dehydrate her somewhat via the intestinal tract, apparently with success.

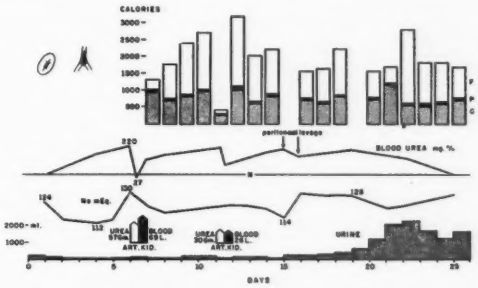

FIG. 2. Course of patient who had embolectomy after mitral commissurotomy, followed by anuria-oliguria. Patient was treated with artificial kidney twice and with peritoneal lavage once. Note adequate caloric intake after the sixth day. *F*, calories derived from fat; *P*, calories derived from protein; *C*, calories derived from carbohydrate.

The patient's hemoglobin had to be maintained at about 8 grams per hundred milliliters with transfusion. Since simple transfusions almost certainly would have overloaded the circulation, 300 milliliters of packed red cells were given while an equal or somewhat larger volume of her own blood was being withdrawn (10), representing a small exsanguinotransfusion.

A temporary urinary suppression is not unusual in patients with mitral commissurotomy. This usually is associated with hypotension and diminished glomerular filtration without actual renal tubular necrosis. The onset of tubular necrosis in such cases is heralded by a decrease of urinary specific gravity that in purely hypotensive cases is normal or high. Once tubular necrosis has become established, restoration of blood pressure does not affect the amount of urinary flow.

ANURIA AND UREMIA IN THE COURSE OF ABDOMINAL SURGERY

Anuria and Uremia After Gastric Surgery

CASE REPORT. A 62 year old man, who, by electrocardiogram, was shown to have an old posterior infarction, had had a subtotal gastric resection and vagotomy for a large duodenal ulcer. At operation, the head of the pancreas was found to be enlarged and infiltrated. On the second postoperative day, the patient developed shock. At reoperation a generalized peritonitis was found. The abdomen was drained in

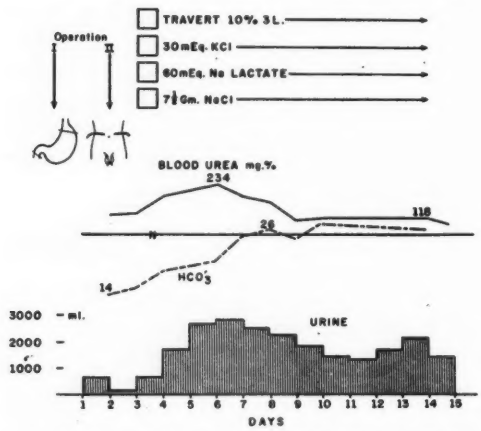

FIG. 3. Course of anuric patient who had peritonitis after gastric resection with multiple drains and short period of oliguria. Three liters of 10 per cent invert sugar (1,200 calories) with electrolytes was given intravenously until patient was able to eat. Note correction of HCO_3'.

three places, and bile drained from the duodenal stump. A high level of serum amylase of 2,000 units indicated pancreatitis. On this day, the patient was almost completely anuric. His alkali reserve was low: 14 milliequivalents per liter. Uremia developed rapidly as the level of blood urea went up to 234 milligrams per hundred milliliters.

Fluid loss was replaced with a continuous intravenous infusion of 3 liters of 10 per cent invert sugar (travert) per day. Acidosis and electrolyte losses were corrected by adding 60 milliequivalents of sodium lactate, 125 milliequivalents (7.5 grams) of sodium chloride, and 30 milliequivalents of potassium chloride (Fig. 3) daily. The blood urea began to fall when the urinary output approached 3 liters per 24 hours. The patient recovered.

The problem of providing calories was less difficult than it is in patients who have anuria without fluid loss. In this case, 3 liters of 10 per cent travert could be used; this provided 300 grams, or 1,200 calories, of invert sugar, per 24 hours.

In planning such a course of infusion it is not sufficient to tell the nurses only that it must run so and so many drops per minute. One must indicate on the bottle with adhesive tape where the fluid level should be at intervals of 2 hours. In this manner, it should be possible to insure a fairly regular infusion rate.

Operation During Undiagnosed Uremia

CASE REPORT. A 65 year old woman had suffered occasional vomiting, severe nausea, and loss of appetite with resulting loss of weight, for almost 1 year. When a nonfunctioning gallbladder containing stones was found, cholecystectomy was performed, and there followed a stormy convalescence. She continued to have severe anorexia and nausea and spells of vomiting, and she failed to respond to medication. She was sent to Cleveland Clinic where she was treated for uremia when a level of over 300 milligrams per hundred milliliters of blood urea was found (Fig. 4). She was given 1 liter of 10 per cent invert sugar with 10 units of regular insulin and 120 milliequivalents of sodium lactate intravenously every day, and 1,800 milliliters of 3 per cent glucose in saline with hyaluronidase (wydase) subcutaneously every other day. After about 12 days, she was able to take a 1,500 calorie, low protein diet with oral electrolyte supplements. She went home much improved but not entirely well, since she has a basic problem of chronic renal and hypertensive disease.

Patients who, like this one, vomit too much to retain oral intake, are given fluids parenterally in the beginning. If part is given intravenously and part subcutaneously, long immobilization with one arm tied is avoided.

Our patient was treated with large amounts of sodium lactate because in the beginning the alkali reserve was very low, under 15 milliequivalents per liter. Correction, or maybe even overcorrection, of the acidosis by rigorous administration of sodium lactate is an important recent gain in the treatment of chronic uremia, as described by Neubauer and Dunsmore (14, 15). The mechanism is not known. Such alkalization may have 2 complications: (1) a steady gain in weight—in other words, the development of edema (as in our patient, Fig. 4); and (2) the development of hypertension with its complications. However, edema and hypertension seem to be less frequent than one would expect from the amount of electrolyte given.

In patients with nausea, anorexia, vomiting, and abdominal pain from obscure causes, such as the one just described and others we have seen, it is well to exclude renal failure as a cause of the symptoms. A striking example was the case of a 60 year old man from whom a normal gallbladder and appendix had been removed. He had continued to vomit

and he was brought to us practically comatose, with a blood urea of 600 milligrams per hundred milliliters. After treatment with the artificial kidney, he was returned to a state of rather benign chronic uremia. He proved to have multiple myeloma with its accompanying renal disease, but is maintaining himself rather well for the 1½ years since the 1 treatment with the artificial kidney.

Anuria (Crush Syndrome) Following Colon Surgery and Peritonitis

CASE REPORT. A carcinoma of the rectum was removed from a 60 year old elevator operator by combined abdominoperineal resection. One hour after operation the colostomy clamp was found to have slipped off so that the open end of the colon had retracted into the peritoneal cavity. The wound was reopened; the colostomy was retrieved; and feces were washed out of the peritoneal cavity with saline. Severe shock was combated with blood transfusions and by winding bandages about each leg. The patient developed peritonitis, icterus, and severe oliguria. Both calves were as hard as wood, and extremely painful; it was believed that the patient had ischemic necrosis of the calf muscles with crush syndrome, caused by tight bandaging of the legs during the prolonged period of low blood pressure.

He received 10 per cent fructose intravenously in a quantity regulated according to the small urinary output and the amount of fluid removed by suction from a gastric tube. Low blood pressures (80/45) were restored to 110/60 almost daily with dextran. The ordinary commercial dextran comes made up in physiologic salt solution. This was the patient's only source of sodium chloride; it sufficed to replace the sodium and chloride losses.

On the fifth postoperative day, the level of serum potassium had risen to 6.3 milliequivalents per liter, for which he was given carboxylic resin sodium salt, 5 grams 12 times per day. On the eighth day, the resin had already caused the level of serum potassium to drop to 4.4; and, as so often happens with resin therapy, it overshot so that later, 125 milliequivalents of potassium chloride had to be given each day (Fig. 5). Blood urea rose to a level of 380 milligrams per 100 milliliters on the eighth day; from then on his uremia began to improve and after a stormy convalescence he left the hospital.

For those who recommend spinal anesthesia as treatment for acute renal failure, it must be discouraging to learn that in this patient the oliguria developed during spinal anesthesia. In certain cases of anuria a spinal anesthesia has been induced high enough to include the kidneys, but we never have noted any benefit insofar as urinary output is concerned.

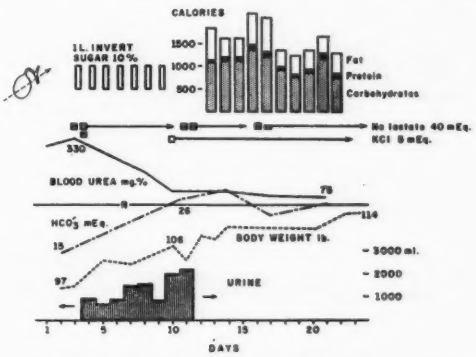

FIG. 4. Course of patient following cholecystectomy during uremia. Low alkali reserve corrected with sodium lactate; after initial intravenous feeding she was able to take high caloric, low protein diet. Note the increase in body weight.

The patient just described had crush syndrome of the lower legs. The term "crush syndrome" recalls to mind the beautiful work of Bywaters during the blitz in London. People that were dug out from under collapsed houses, who had had their legs crushed by debris, developed anuria, and they died after some days—even though they had survived the initial period of shock. Bywaters relates the story of one man who was covered by debris that severely crushed his legs while he was drinking beer in a pub. This man did not develop anuria. This experience induced Bywaters to add a kind of riding canteen to his emergency squad so that he could administer all types of drinks through rubber tubing to people covered with debris. At that time, it was believed that the administration of fairly large amounts of fluid and alkali might prevent the precipitation of hemoglobin or myohemoglobin in the renal tubules, and thereby prevent anuria not only after crushing injury, but also after mismatched transfusion.

The recent demonstration by Oliver and associates that renal failure in such cases is primarily attributable to an episode of renal ischemia with resultant necrosis of tubular

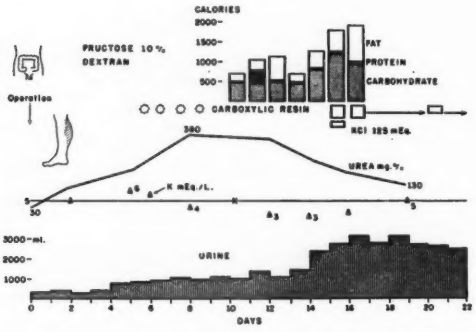

FIG. 5. Course of patient who had crush syndrome following hypotension and bandaging of legs during shock caused by peritonitis after abdominoperineal resection of the rectum.

cells and disruption of basement membranes lessens the belief in this form of therapy. However, pigmentary plugs may be additive to ischemic injury and I have seen favorable results in several patients with hemolytic reactions, after the immediate administration of 2 liters of 5 per cent glucose and 500 cubic centimeters of 5 per cent sodium bicarbonate. If it is not given immediately, but after the damage already has been established, fluid and alkali are useless and carry the risk of overhydration and alkalosis.

The use of resins to remove potassium in patients with acute renal failure has been advocated by Elkinton and associates. The cation exchange resins are prepared in different cycles, as the sodium salt, the hydrogen cycle, and the potassium salt. The resins used to treat edema are sodium-binding because they are mixtures of the potassium salt and the hydrogen cycle (Fig. 6). In hyperkalemia we want the sodium salt, 20 to 60 grams per day in divided doses. This resin will exchange its sodium for potassium. The hydrogen cycle may be used, but it will take out both sodium and potassium in exchange for hydrogen ions, with resultant acidosis.

In patients with uremia who require a plasma expander, dextran should be used in preference to plasma itself, and on many occasions in preference to blood, because dextran, when it is not excreted, finally is metabolized to carbon dioxide and water. It does not form nitrogenous waste products that for ultimate removal depend upon the kidneys; whereas, plasma, and blood break down to urea and will aggravate the uremia. Dextran as a carbohydrate provides some calories and may have a protein-saving effect. The difference between the effects of blood and dextran on the blood urea may be demonstrated (although, of course, this is no proof) by a comparison of the graphs pertaining to 2 patients with uremia (Fig. 7).

CASE REPORTS. These 2 patients were seen in Holland during a period of time in which we saw many cases of glomerulonephritis and some of these cases were associated with hypotension. Each patient required plasma expanders to maintain blood pressure. For the one patient, treated in 1947, blood was used. This blood was broken down to urea; the level of blood urea went up and up and the patient died in uremia. The second patient, who was treated in 1948, had a comparable rate of urea excretion; however, dextran was given; the level of blood urea did not increase, and she recovered.

In patients who require plasma expanders for maintenance of blood pressure while they are anuric, dextran is preferable to other substances such as blood or plasma, because it metabolizes without placing any demand on the kidneys.

Hepatorenal Syndrome

This heading loosely includes a great variety of clinical conditions. These vary from acute glomerulonephritis with incidental icterus—for which the prognosis is quite good—to severe hepatocellular damage with yellow atrophy and renal tubular necrosis—for which the prognosis is grave. Icterus following surgery of the liver or bile ducts, or infectious cholecystitis, quite frequently is accompanied by some degree of renal tubular damage, and sometimes by a hepatorenal syndrome. Recently, 4 of our patients with severe hepatorenal syndrome recovered after treatment with the artificial kidney. One of these cases has been reported in a previous article (11).

In the following case of a hepatorenal syndrome, the patient had icterus with a dis-

turbed electrolyte balance, which led to uremia.

CASE REPORT. A 76 year old woman had had stricture of the common bile duct; continuity with the duodenum had been restored after 2 operations, but severe biliary cirrhosis had developed. On 2 occasions I was asked to see her while she was in poor condition with hyponatremia. The serum sodium level was 105 on one occasion and 113 milliequivalents per liter on the other, with blood urea levels respectively of 157 and 186 milligrams per hundred milliliters. The serum sodium was restored to about 130 milliequivalents with immediate improvement.

The patient left the hospital reasonably well, with a blood urea value of 36 milligrams per 100 milliliters and with only insignificantly small leaks from a biliary fistula.

In this case the uremia should be considered as *urémie par manque de sel* (uremia by lack of salt). Figure 8 shows the nomogram of Wolf and McDowell used to calculate sodium replacement.

We do not replace sodium in patients having acute anuria, unless the level is extremely low, or when sodium is demonstrably being lost, e.g., from fistulas or from the gastrointestinal tract.

Uremia in Pancreatitis

During the last few years, we have seen many cases of pancreatitis; sometimes unrecognized before operation, sometimes apparently aggravated or produced by operations on the bile ducts or in the region of the pancreas, and once caused by steering-wheel trauma to the upper abdomen.

According to Lynch, the renal lesion is at least partly due to fat embolism, originating from fat necroses. The fat emboli first block the glomerular capillaries and later move on to the peritubular circulation.

The amount of urine is not as much depressed as in many other conditions of acute renal insufficiency and may be 500 to 700 cubic centimeters per day; but, on the other hand, the specific gravity is low and there is no tendency to improvement. In the cases of hemorrhagic pancreatitis, hypotension tends further to depress the urinary output.

We have used artificial hibernation in 2 patients desperately ill with pancreatitis. We

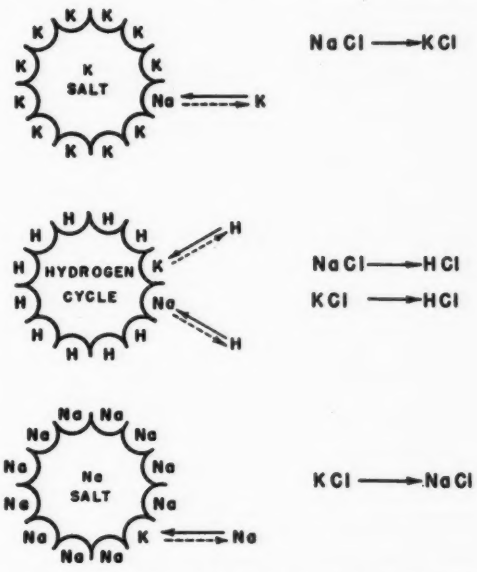

FIG. 6. Mechanism of carboxylic resin cation exchangers.

followed the French technique described by Huguenard and Laborit as closely as possible, and it is our opinion that in each case hibernation was responsible for prolonging life.

CASE REPORTS. A 34 year old man had an automobile accident, suffered multiple fractures and bruises, and developed pancreatitis probably as a result of steering-wheel trauma. He was almost moribund when hibernation was started and he lived for 6 days thereafter. He awoke several times, was able to speak, but his azotemia progressed to 500 milligrams of urea per 100 milliliters of blood, and had to be reduced with the artificial kidney. He finally died from massive pulmonary embolism.

The second patient, a 58 year old woman, survived a 3 day hibernation that interrupted a downhill course of septic temperatures and repeated collapse. She slowly improved, attained a normal level of blood urea but, 19 days after the termination of the hibernation, she died from cardiac arrest while sitting in a chair. At postmortem examination, a large torpid retropancreatic abscess was found.

Hibernation may have something to offer to patients with pancreatitis when all other methods fail. Dialysis will effectively but temporarily lower the uremia.

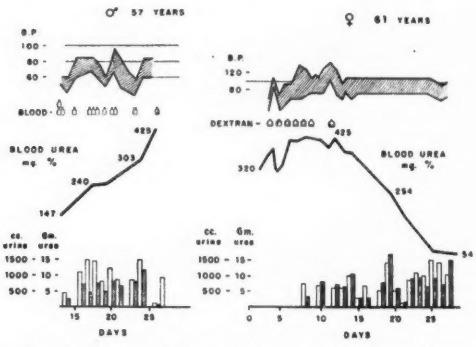

FIG. 7. Courses of 2 anuric patients with glomerulonephritis and equal daily urea excretions. Note rise in blood urea level of patient who was transfused; no rise in patient who was given dextran.

ANURIA AND UREMIA COMPLICATING GYNECOLOGIC SURGERY AND OBSTETRICS

Anuria Following Septic Abortion

CASE REPORT. In a 39 year old woman a hysterectomy was performed for an infected abortion. Peritonitis and septic temperatures were treated with antibiotics. Severe oliguria (less than 150 cubic centimeters per day) developed and persisted for 11 days. She was treated twice with the artificial kidney; on both occasions her clinical condition improved. Because of the peritonitis she was unable to retain anything by mouth, so she was given hypertonic (10 per cent) invert sugar (travert) subcutaneously with hyaluronidase (wydase) (Fig. 9). She went through a temporary psychosis but recovered completely from this and from the renal disease. Unfortunately, an unstable gait, due to vestibular injury, has persisted for more than a year.

The damage to the vestibular system was probably caused by streptomycin or dihydrostreptomycin which could not be excreted during the period of anuria. In one's attempt to relieve the most serious condition first, such as the peritonitis in this case, it sometimes is forgotten that later complications may become very embarrassing. Streptomycin should be avoided if at all possible during acute renal failure.

While in the old times we used to think of the uremic patient as one who is sleepy or comatose, now, more and more we see patients who are not sleepy, but, rather, who are intensely restless, paranoid, or hysterical. The patient just described is an example. Before and after her illness she had a most stable, pleasant, and charming personality. During severe uremia and in the diuretic phase, she developed a hysteric paranoid state with a most severe ataxia, to which latter the vestibular damage may have contributed.

It is surprising that 10 per cent invert sugar in water can be administered subcutaneously if hyaluronidase is added. In an occasional patient it has caused some discomfort.

Anuria Following Retroplacental Hemorrhage

CASE REPORT. A 38 year old woman with known hypertension (200/100) had a renal shutdown following retroplacental hemorrhage. She was treated with potassium-binding resin and a forced high caloric, low protein diet supplemented with a commercial fat emulsion, ediol. The blood urea went up to 210 milligrams per hundred milliliters but, on the sixth day, the urinary output increased to 900 milliliters. Fluid intake was regulated according to the patient's daily weight. During the diuretic phase, 5 grams of sodium chloride and 1 gram of potassium chloride had to be administered to avoid depletion of sodium and potassium (Fig. 10). The patient recovered and was restored to a state of benign hypertension such as had existed before her pregnancy. One year later she reported that she was doing well and was able to take care of her household.

Note that this patient was not given a single intravenous or other infusion during her stay in our hospital.

A retroplacental hemorrhage often occurs on the basis of some degree of toxemia of pregnancy, which in itself may lead to eclampsia and posteclamptic anuria. This patient may have had symmetrical cortical necrosis. Gradual transition certainly occurs from the more common tubular necrosis to what is called symmetrical cortical necrosis, usually, but not exclusively, seen after accidents such as a retroplacental hemorrhage. The prognosis is not always hopeless and pre-existing hypertension need not preclude recovery from acute renal insufficiency.

Anuria Following "Pelvic Sweep"

If pelvic exenteration is complicated by oliguria or anuria, it may be quite difficult

to ascertain whether this is due to tubular necrosis or to damage, kinking, or encroaching upon the ureters. When fluid, which may be urine, is obtained from the colon or the Bricker pouch into which the ureters are transplanted, a high level of urea therein indicates partial obstruction and excludes tubular necrosis. In a recent experience where a high urea (1,400 milligrams per hundred milliliters) was found in the fluid, changing of the patient's position on the operating table prior to an intended ureterostomy suddenly released a flow of urine.

UREMIA AND GENITOURINARY SURGERY

Uremia Caused by Nephrolithiasis

In a patient with chronic uremia, an operation must be postponed when at all possible until medical management has corrected electrolyte imbalance and reduced blood urea. This often can be accomplished. The artificial kidney sometimes is used to reduce uremia prior to surgery; so far I have not needed it under such circumstances.

CASE REPORT. A 37 year old housewife was admitted for colic and hematuria due to nephrolithiasis; a left nephrectomy had been performed 7 years previously for stones. Prior to admission, she had become progressively uremic, with severe headache and vomiting. The serum calcium was low, 8 milligrams per hundred milliliters; and the serum phosphorus was high, 6.7 milligrams per hundred milliliters. The blood urea was 168 milligrams per hundred milliliters, and the alkali reserve was 9 milliequivalents per liter (Fig. 11).

The low alkali reserve was an indication that she should be treated with sodium lactate; the high phosphorus level that she should be given aluminum hydroxide by mouth, in the hope that it would bind phosphate in the intestinal tract and thus would allow the phosphate level to fall and the calcium level to rise. The dosage of aluminum hydroxide was 5 or 10 cubic centimeters every hour or 2 as tolerated.

In the beginning, parenteral fluid and carbohydrate administration was necessary on account of vomiting, but soon a forced high caloric (20 to 30 grams of protein) diet was given; the composition of the diet is shown in the blocks on the top graph in Figure 11. For this particular patient the diet was high in carbohydrate, low in protein, and also low in fat because of gallbladder complaints. After the blood urea level had been kept normal for 12 days, the renal stones were removed. The patient required much care after the operation, but she left the hospital greatly improved.

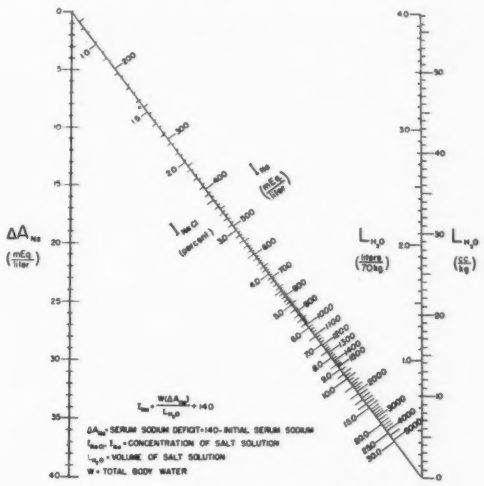

FIG. 8. Nomogram of Wolf and McDowell for sodium replacement, by permission of the authors, and the editor of *Journal of Applied Physiology* (18). If there is a deficit of 25 milliequivalents per liter between the patient's level of serum sodium and a normal level of 140, with a ruler you connect the 25 on the left vertical scale with the 1 liter point on the right vertical scale (assuming that the patient's weight is 70 kilograms); then you will find on the sloping scale in the center the total of 1,200 milliequivalents of sodium that must be replaced, which would amount to 7 per cent if given in 1 liter. If one wishes to give it in the form of 3 per cent saline solution, one connects the 25 mark on the left vertical scale with the 3 per cent point on the sloping scale, and finds on the right vertical scale that 40 cubic centimeters per kilogram of body weight of 3 per cent saline is to be given. Needless to say, it is extremely dangerous to correct all at once a large deficit of sodium in a patient who has become accustomed to a low level of sodium. One should allow 2 or 3 days at least for effecting the corrections, and as a rule they should be made only after diuresis has been established.

We believe that Borst was right when he found that a high caloric, low protein diet, whenever possible, was of great benefit not only in reducing chemical uremia, the levels of urea, creatinine, uric acid, and other retention products in the blood, but also in greatly improving the well-being of the patient. As the patient really is not only persuaded but almost compelled to eat his diet, the name "forced" high caloric, low protein diet is appropriate (7). The blood urea of a

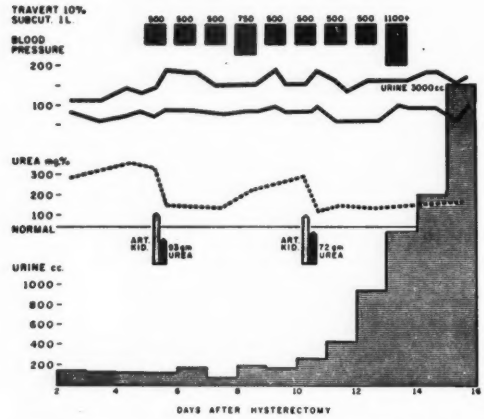

FIG. 9. Course of anuric patient who had acute tubular necrosis following hysterectomy for septic abortion with peritonitis.

patient with moderate chronic uremia nearly always can be reduced to a normal or slightly elevated level with this diet and with control of electrolytes, especially with sodium lactate.

Kidneys, even after prolonged periods of obstruction, are able to recover function in an amazing way after removal of the cause of the hydronephrosis.

Uremia Caused by Hypertrophy of the Prostate

This form of obstructive uremia usually subsides promptly with drainage and provision of fluid and electrolyte. Sometimes a patient may require very large volumes of fluid.

CASE REPORT. A 65 year old man had gastrointestinal symptoms that appeared to be caused by uremia. He was found to have benign hypertrophy of the prostate with a residual volume of urine of 800 milliliters. The bladder was drained by an indwelling catheter. Although the electrolytes were not greatly disturbed, the blood urea went up to 264 milligrams per hundred milliliters—despite a urinary output of 3 liters per 24 hours, which usually is adequate. We decided to administer large amounts of water by all possible routes. When the urinary output had reached 8 liters per day, the blood urea level began to fall. It reached 107 milligrams per hundred milliliters, below which level it would not drop. The further course was complicated by attacks of Stokes-Adams syndrome. He left the hospital with the indwelling catheter still in place, instructed to maintain a water intake of 3 liters daily and to provide against salt loss by taking 5 grams of salt capsules daily.

This is rather an unsatisfactory result. Still, the patient's condition improved markedly when 8 liters of fluid were administered per day. Fluid may be administered in the following ways: (1) by continuous gastric drip, 2,400 milliliters; (2) intravenously—10 per cent invert sugar, 2,000 milliliters; (3) subcutaneously with hyaluronidase, 1,800 milliliters; or (4) by mouth, 2,000 milliliters. Sodium lactate and sodium chloride and potassium chloride may be added to each of the infusions. Commercial fat emulsions, ediol or lipomul, and sugar may be added to the gastric drip.

Thus, we must give some patients more fluid than common sense would suggest, others must have much less than seems reasonable—as in the next case, presented here, although he was not a genitourinary surgical patient.

CASE REPORT. A gastrojejunocolic fistula was closed in a 40 year old man. He was extremely emaciated. The hands were purple with marked vasodilatation; he had a tendency to develop a low blood pressure, which had been ineffectually treated with large amounts of glucose and electrolyte solutions intravenously. The most marked symptoms were: constant gross twitching and hyperkinesis. Both these symptoms disappeared promptly after the large volume

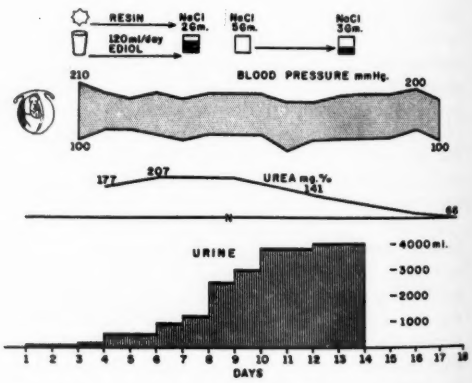

FIG. 10. Course of anuric patient who had symmetrical cortical necrosis (?) following retroplacental hemorrhage.

intravenous therapy was replaced by the administration of dextran and a relatively small volume of 10 per cent travert, the former to maintain plasma volume and the latter to provide some caloric intake.

Anorexia, nausea, and vomiting are uniformly present in uremia. The vomiting may persist uncontrollably even when blood urea has been decreased. The associated anorexia seriously interferes with feeding. Chlorpromazine (thorazine) often is helpful to suppress nausea and vomiting; however, some patients with uremia, when given chlorpromazine in amounts even as small as 25 milligrams 3 times per day may go into an extremely quiet state, reminiscent of hibernation even though the temperature is not low. They become apathetic; they will stop eating and drinking if left alone, so that the blood urea level will increase. This desuetude that may become a coma is not due to uremia alone, but to chlorpromazine in the presence of uremia. Phenergan may have a similar antiemetic action and, although structurally related, it does not seem to cause hepatic damage, such as chlorpromazine does in rare instances.

Anuria Following Intravascular Hemolysis During Transurethral Prostatectomy

When water, used to distend the bladder, enters into the veins of the prostatic bed during transurethral prostatectomy, intravascular hemolysis follows. Bronchopneumonia often has been the final complication in old people. Such bronchopneumonia may be symptomless and afebrile and therefore treacherous.

ANURIA FOLLOWING BLOOD TRANSFUSION AND OTHER HEMOLYTIC ACCIDENTS

Incompatible transfusions will continue to occur as long as blood transfusions will be given and, since large amounts of blood often are given during or after operation, surgeons will continue to see transfusion reactions. They are more dangerous in the more seriously ill patients. The possibility of other hemolytic accidents is not sufficiently realized.

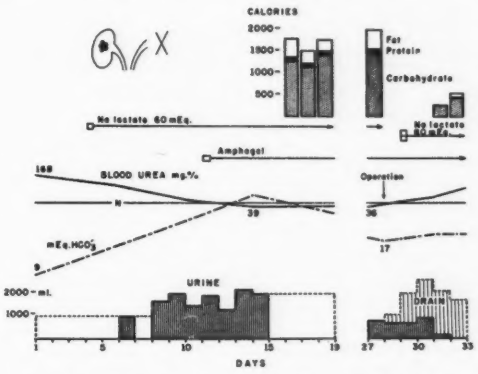

FIG. 11. Course of patient who had nephrolithiasis and uremia. Patient was treated with sodium lactate; aluminum hydroxide; and high caloric, low protein diet, prior to surgery.

Hemoglobinuria Following Administration of Distilled Water Intravenously

Intravascular hemolysis may occur if a patient accidentally is infused with distilled water instead of isotonic electrolyte or glucose solution.

CASE REPORT. A 60 year old man had had a vagotomy and retrocolic gastroenterostomy for obstructing duodenal ulcer. After operation he continued to be obstructed and he was maintained with intravenous fluids. Accidentally, he was given 1 liter of distilled water instead of 5 per cent glucose intravenously, which was followed by a 1½ day period of hemoglobinuria. He immediately was given 100 cubic centimeters of 25 per cent mannitol and 2 liters of 5 per cent glucose with water and there was no urinary suppression. The urine, which in the beginning had the color of Burgundy, gradually cleared.

Mannitol was given immediately after the accident that produced hemoglobinuria, in the hope of preventing renal shutdown by producing osmotic diuresis. While there seems to be little against its use, unfortunately its effectiveness has not been proved in patients. In "crushed," myohemoglobin-treated rats, prompt osmotic diuresis decreases renal damage.

Anuria After Subcutaneous Administration of Blood

Recently a patient was anuric for 6 days, who had received subcutaneously 1 pint of

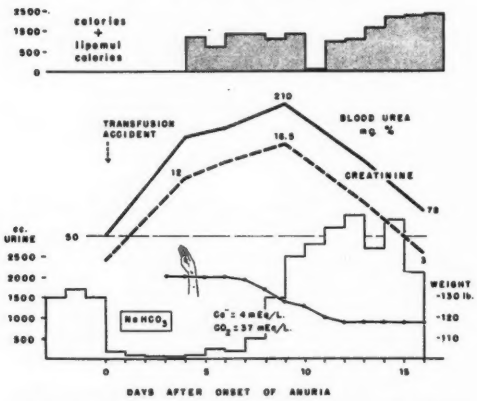

FIG. 12. Course of patient who had anuria after transfusion accident and was erroneously (too late) given sodium bicarbonate, resulting in tetany; otherwise management by diet only.

blood presumably of the correct group. The pathogenesis of this anuria is even more obscure than that of the usual transfusion accident.

Anuria After Transfusion of Incompatible Blood

CASE REPORT. A 30 year old woman had been given a transfusion in a small hospital after a normal delivery, for the only reason that she "looked a little washed out." The transfusion had been continued in spite of chill and back pain. She developed anuria. On the second day she was seen by an internist who recalled something about sodium bicarbonate and gave her large amounts of sodium bicarbonate on the second and third days, at a time when she was already anuric. The result was an alkalosis with a HCO_3' level of 37 milliequivalents per liter. She was transferred to Cleveland Clinic Hospital.

Although serum calcium was 4 milliequivalents per liter (8 milligrams per hundred milliliters) she developed pain and tingling in the hands and feet, which gradually progressed to tetany. After a single injection of 1 gram of calcium gluconate, the tetany and pain disappeared (Fig. 12).

She was further treated with a forced high caloric, low protein diet and fluid restriction. Diuresis started on the eighth day and she recovered after a short period of hysteria.

This is the second patient in this series who did not receive a single intravenous infusion on our service.

Alkalosis decreases the ionizable calcium, so that, for a given serum calcium, tetany occurs sooner in alkalosis than in acidosis. The alkalosis was not treated with hydrochloric acid intravenously or with the hydrogen cycle of carboxylic resin orally, as the acid that accumulated during anuria would in time compensate for the excess of sodium bicarbonate. Indeed, no more attacks of tetany occurred.

It is quite distressing to see how many blood transfusion reactions have occurred in patients who really did not need transfusion. Blood transfusion carries a morbidity and mortality equal to some surgical procedures. I have seen another young woman who developed anuria, lasting 10 days, after she had been given a mismatched transfusion for the same reason: she was pale after a normal delivery. A few iron pills and a little patience would have cured this anemia (if it existed) without any risk of reaction.

Too often the blood transfusion is not discontinued when a reaction sets in. Most distressing is the following case. A doctor's wife had a severe chill while receiving a blood transfusion. Her husband came in, shut off the blood and left the room to find the surgeon. An intern came in, turned the blood open and infused the rest of it. She died 15 days later in anuria.

Uncomplicated transfusion anuria often can be managed without the aid of dialyzing methods. However, if a transfusion reaction does occur in patients that already are severely ill from other reasons—operation, trauma, infection, toxemia of pregnancy, hepatic failure—and anuria supervenes, then the superimposed uremia may make the condition irreversible unless dialysis is employed. In some instances, patients may die before dialysis is indicated.

CASE REPORT. A 55 year old man with a long foreign name had had grafts of the lower aorta and of both iliac arteries. During operation, he had received 7 pints of blood that had been cross-matched and typed not with his blood, but with the blood of another man on the same floor, who had a similarly long foreign name. Anuria developed, notwithstanding the high spinal anesthesia that had been used during the operation and that had been continued after it in the hope of preventing renal shutdown, and notwithstanding the administration of 2 liters of 5 per cent

glucose and mannitol. He died 3 day later in cardiac failure.

By the time the error had been clarified, it was too late to give an exsanguinotransfusion; this might have prevented the effects of the circulating mismatched blood. As exsanguinotransfusion is performed in France as described by Tzanck and associates, the patient's blood is removed from one side while fresh blood is infused in the other side (10, 17).

The most common reason for incompatible transfusion in large institutions is not a technical error in typing the blood, but is a clerical error. In retrospect, I believe that I personally made the mistake of being satisfied when I was informed that the blood had been rechecked and had been found to be compatible. What I should have done, and have done since in every questionable case, is: draw blood from the patient myself; take it down to the blood bank myself; see the hemolysis myself; and have the plasma crossmatched before my eyes with cells from the incriminated bottles.

CONCLUSION

Surgery or trauma may lead directly to certain forms of renal failure, and surgery may provoke or may coincide with accidents that lead to renal insufficiency. Coronary thrombosis, thrombosis of arteries to legs, mesentery, or kidneys, subarachnoidal hemorrhage, hepatorenal syndrome, pancreatitis, infusions of distilled water, mismatched transfusions may happen during or shortly after surgery, and each condition may cause renal failure.

Very often renal shutdown develops on the basis of pre-existing renal disease. It may be impossible to unravel the etiology for a given clinical situation, and until proved otherwise, one has to treat the patient in the hope that he has a curable disturbance of renal function.

An outline of the treatment of acute and chronic uremia in surgical patients, as it has been presented in the preceding pages, follows.

SCHEMATIC SUMMARY OF PRESENT-DAY MANAGEMENT OF ANURIA AND UREMIA IN SURGICAL PATIENTS

Attempts to Prevent Acute Tubular Necrosis

1. Restoration of low blood pressure either at the onset of acute anuria or in its later course. This is best done with 6 per cent dextran in 5 per cent glucose (not in saline as most of the commercial dextrans come). Dextran is better than blood, unless blood is demonstrably lost. The virtues of restoring renal function with pressor substances like noradrenalin are still open to speculation, and these do not replace blood volume in oligemia.
2. In cases of hemoglobinuric (incompatible blood) or myohemoglobinuric nephrosis (crush syndrome), perhaps of use if immediately administered:
   A. 2,000 milliliters of 5 per cent glucose plus 500 milliliters 5 per cent $NaHCO_3$ I.V.* (*Dangerous if given when kidneys already are blocked.*)
   B. Osmotic diuretic: 100 cubic centimeters 25 per cent mannitol I.V. (Other diuretics are dangerous.)
   C. Large exsanguinotransfusions to remove free hemoglobin.
   D. All other methods: intermittent high spinal anesthesia, procaine I.V., and decapsulation seem to be either useless or harmful.

In the Presence of Renal Failure

Once renal failure is an established fact the measures mentioned are only harmful. For further management it is important to recognize whether a patient is in an anuric (or severely oliguric) phase, or in a diuretic phase.

Most patients with chronic uremia must of necessity excrete large amounts of urine and to those cases, the recommendations listed under chronic uremia pertain. Some, however, are oliguric, notwithstanding sufficient hydration; they should be treated as in acute anuria.

1. Maintenance of body fluid.—Acute anuria: anuric phase—700 milliliters per day plus compensation for loss; diuretic phase—enough to prevent dehydration, 2,000 or 3,000 milliliters per day. If overhydrated during anuric phase: venesection, Na sulfate 50 grams per os to cause diarrhea; filtering type of artificial kidney (most effective), or dialysis with dialyzing fluid that is made hypertonic by addition of glucose to its usual composition, either in artificial kidney or in peritoneal lavage.
   Chronic uremia: fluid often has to be forced, sometimes with NaCl.
2. Control of electrolytes.—Dialysis will correct the hypotonic or hypertonic status of any electrolyte within a few hours; avoid restoring all Na^+ at one time.

*I.V. = intravenous(ly).

Na^+	Acute anuria:	No Na^+ administration; compensation for loss only (see exception under "Acidosis").
	Chronic uremia:	Na lactate 120 milliequivalents per day to restore HCO_3'. NaCl to replace urinary NaCl loss. Rarely salt-free diet when indicated by hypertension, edema, or cardiac failure.
K^+	Hyperkalemia (often in acute anuria):	Carboxylic resin sodium salt administration (30 to 60 grams per day in divided doses, with care against overshooting; give laxative with it). Hypertonic glucose (plus insulin) or invert sugar I.V. High caloric, low protein diet. If serious, dialysis.
	Hypokalemia (often in chronic uremia or in diuretic phase of acute uremia):	Administration of KCl, slowly I.V. (20 milliequivalents mixed in large volume diluent) or K gluconate (kaon) by mouth.
Ca^{++}	Often hypocalcemia, but not so often tetany:	1 gram Ca gluconate I.V. Aluminum hydroxide, to bind P, multiple doses as much as patient can take (5 to 10 milliliters 12 times per day).
	Acidosis—acute anuria or uremia:	Carbohydrate. Rarely Na lactate (40 milliequivalents) or Na bicarbonate (4 grams per day), only if HCO_3' is lower than 12 milliequivalents per liter. Dialysis, which removes the abnormal acids.
	Chronic uremia:	Na lactate 120 to 180 milliequivalents per day; K gluconate if K^+ also is low. Rarely (but sometimes) dialysis.

3. Suppression of protein catabolism with high caloric, low protein diet.—Avoid transfusions and surgery if at all possible; they increase protein catabolism. Forced high caloric (over 2,000 calories), low protein (20 to 40 grams) regimen (Borst) is very useful in chronic uremia; in acute uremia, administer as much as is practical. Keep diet diary and calculate caloric intake. Try fat emulsion (ediol, lipomul oral) by mouth or (diluted) by gastric drip. If necessary: 20 per cent invert sugar (travert) plus insulin I.V. or hypertonic (up to 40 per cent) glucose or invert sugar by cardiac catheter. Intravenous fat emulsion (may be practical in the future).
4. Prophylaxis and treatment of infections with antibiotics.—Erythromycin seems to be well tolerated. Streptomycin, when not excreted, may accumulate and cause irreparable vestibular (nerve) damage.
5. Elimination of retention products by dialysis.—Also removal of acid metabolites, correction of electrolytes, and reduction of edema.

Artificial kidney:	5 to 6 hours of dialysis will suffice, and its effect lasts 4 to 7 days.
Peritoneal lavage:	not often possible in postoperative patients.
Intestinal lavage:	electrolyte regulation difficult.

Unit-Conversion Tables

1 mEq. NaCl	=24+34	= 58 mgm.
1 mEq. $NaHCO_3$	=24+1+12+48	= 85 mgm.
1 mEq. Na lactate	$=CH_3CHOHCOONa$	=112 mgm.
1 mEq. KCl	=39+35	= 74 mgm.
1 mEq. $KHCO_3$	=39+1+12+48	=100 mgm.
1 gm. NaCl	=17 mEq.	
1 gm. $NaHCO_3$	=12 mEq.	
1 gm. KCl	=13 mEq.	
1 gm. $KHCO_3$	=10 mEq.	

To make a solution of Na lactate of approximately 1 mEq./ml. take ½ pound (280 c.c.) commercial Na lactate solution (70 to 80 per cent) and add water to make 1 quart. This may be taken by mouth 30 ml. t.i.d.

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A 13 YEAR STUDY OF ACUTE PANCREATITIS

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ACUTE PANCREATITIS is a disease which merits continuing study in regard to its diagnosis, natural history, and therapy. It produces a clinical picture which often suggests acute cholecystitis, perforated peptic ulcer, acute bowel obstruction, or vascular accident of the abdomen. As a result, patients are subjected to an emergency operative procedure when, if the true diagnosis were known, operation might be avoided or at least postponed until specific indications arose. It has been found that acute interstitial pancreatitis usually subsides quickly and spontaneously with nonoperative therapy. Acute hemorrhagic pancreatitis, however, places the patient in an unpredictable situation. Here, unnecessary operation may tip the balance and cause death when recovery might otherwise have been possible. In certain other patients operation may be the only way of saving life. Thus, it is seen that it would be a valuable asset to have the ability to: (1) differentiate pancreatitis from the other abdominal crises, and (2) differentiate the hemorrhagic and necrotic type from the interstitial variety. A careful study should be made of a statistically significant number of cases with proved diagnosis in order to become familiar with the clinical picture produced and the outcome of the disease. At the same time a study of diagnostic aids and therapy may be made. Several good reviews of the disease have been written in recent years, but it is believed that there is still information to be gained from studies of groups of patients, and for this reason the following investigation was undertaken.

This series of 74 patients includes those patients admitted to the hospital between 1941 and 1954 in whom a diagnosis of acute pancreatitis was made by one or more of the following: (1) a significant serum amylase elevation, (2) a positive urinary amylase in 1 case, (3) operative findings, and (4) necropsy. It is interesting to note the difference in incidence of the diagnosis through the years. From 1941 through 1949 there were 37 cases proved, while from 1950 through early 1954 already there have been another 37 cases proved (Fig. 2). Perhaps a reason for this is the increased literature regarding the disease, which has made physicians more aware of its occurrence, plus the increasing use of serum amylase determination in differential diagnosis of the acute surgical abdomen.

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VITAL STATISTICS

At this hospital the subjects of acute pancreatitis have been predominantly white (63 as compared with 11 nonwhite) and have been more frequently male (46 as compared to 28 females). Twenty-three of the patients have been obese. As shown in Figure 1, there is a peak incidence of acute pancreatitis in the younger age groups, with the maximum in the third decade, and thereafter a steady decline. This information does not agree with a report by Fallis in 1951. In a study of 250 cases he found that the highest incidence was in middle age, females were more prone to have the disease, and the body weight was usually over 160 pounds.

In the present series the hospital mortality has been 16 per cent, which coincides fairly closely with studies by Raker and by Siler and their associates. All of the deaths have been due to acute hemorrhagic necrosis, except in 1 case of acute suppurative pancreatitis and 1 of acute interstitial pancreatitis complicated by diabetes. Of the patients with proved hemorrhagic necrosis 7 were over and 4 under 50 years of age.

HISTORY

Of the 74 patients, 14 (18.5 per cent) did not present a past history relevant to the disease. In the remaining 60, the most frequent background was a history consistent with chronic cholecystitis, namely fatty food intolerance, "indigestion," excessive gaseousness, or recurrent bouts of right upper quadrant pain. The next most frequent item revealed in the history was chronic alcoholism. Aside from these, 6 patients had had an onset of infectious parotitis 5 to 7 days previously, and 1 a viral hepatitis the preceding year. In 3 cases, the attack of acute pancreatitis was a postoperative complication (1 on the fifth day after colostomy for carcinoma of the sigmoid, and 2 on the fourteenth day following cholecystectomy). Acute pancreatitis occurring as an immediate postoperative complication is seen frequently enough to have stimulated investigations toward an appropriate explanation, but so far the answer remains hidden. Operative procedures in which there has been considerable trauma may have little or no effect on the pancreas, while others associated with very little trauma have resulted in stormy postoperative courses because of pancreatitis.

SYMPTOMS

The complaint of epigastric pain is almost invariable. Frequently, however, the pain is described as upper abdominal; in 10 per cent the lower quadrants are the sites of pain. The pain radiates straight through to the back in only 26 per cent of the patients, and radiation to the shoulders or scapular areas is very infrequent (5 per cent).

Nausea and vomiting are almost always present. Constipation and diarrhea occur with equal infrequence, and chills have often been reported.

PHYSICAL SIGNS

Abdominal tenderness was always demonstrated except in comatose patients. Muscular guarding, however, was presented by only 52 per cent, and a board-like abdomen is almost rare. Cattell and Edlin and their associates and Fallis too, have made note of this lack of parallelism of muscle guarding to the degree of subjective pain. Rebound tenderness was present in 41 per cent and ileus was noted in 29 per cent of the cases on the first examination. Both of these findings frequently developed after admission to the hospital in cases in which they were not present initially.

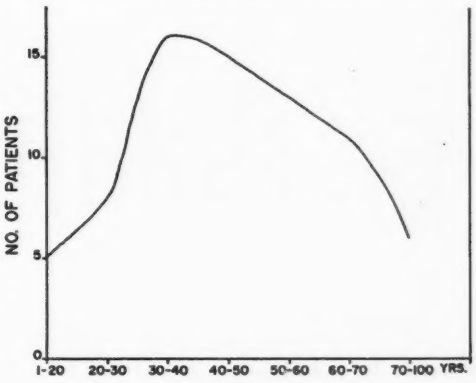

FIG. 1. Age incidence.

The temperature was elevated in 48 per cent of the cases. Only 8 patients were admitted in a state of shock. In agreement with other writers on the disease, shock has been a bad prognostic sign. Jaundice was seen in 10 patients and was of transitory nature in all of these.

Roentgenographic studies are of recognized but limited value in the diagnosis of acute pancreatitis. A widening of the duodenal curve indicating edema of the pancreas, or distention of an isolated loop of jejunum in the left upper quadrant, or both, are suggestive of acute inflammation in the pancreatic area. In the present series, the "sentinel loop" of jejunum was not a frequent finding. Usually there was indication only of generalized ileus or a left upper quadrant mass. In only 7 of the 74 cases was such roentgenographic evidence present as to suggest acute pancreatitis. This figure excludes those films demonstrating choleli-

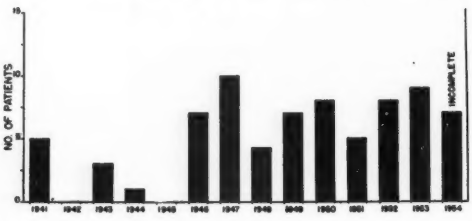

FIG. 2. Number of cases per year.

thiasis, since from them no differentiation can be made between acute gallbladder disease and acute pancreatitis.

LABORATORY FINDINGS

A significant degree of leucocytosis (11,000 or over) was present in 52 of the 74 cases (69 per cent), and the serum amylase was elevated in 71 per cent of the patients, or 83 per cent of those on whom the test was performed. Of the 22 patients who did not show an elevated serum amylase, 2 were practically moribund when the blood was drawn, in 14 no amylase determination was done, and the other 6 were in the subsiding phase of the disease as was indicated by falling values on successive days or by the history. The value of serum amylase determinations in diagnosis of this disease is well recognized today. Though it can be elevated in other diseases, such as perforated peptic ulcer, small bowel obstruction, acute peritonitis, and renal insufficiency, the elevation in these instances does not usually appear until the fifth to the fourteenth day and does not interfere, therefore, with the diagnosis of the acute abdomen. Patients sensitive to codeine or morphine can show an elevation of serum amylase 2½ to 5 hours following injection of these drugs but such instances are infrequent. Amylase determinations of urine and peritoneal fluids are also of value as diagnostic aids. Only 2 patients had lowered serum calcium values. One had a value of 7.29 milligrams per cent the first day after cholecystostomy, with a rise to 9.5 milligrams per cent on the third postoperative day; the patient made an uneventful recovery. Another patient had a serum calcium of 8.76 milligrams per cent and recovery was complicated by the formation of a pancreatic abscess. The degree of hypocalcemia seems to parallel the amount of fat necrosis, and the calcium is probably removed from the serum store by precipitation with fatty acids from the necrotic fat, as well as by excessive excretion into the bowel under the influence of increased adrenal cortical stimulation. Serum calcium levels of 7 to 8 milligrams per cent indicate very severe degrees of pancreatitis, and those patients with a calcium level below 7 milligrams per cent have a grave prognosis. In other studies of pancreatitis, the calcium level has been found to be at its lowest during the second to the fourth day of the disease.

TREATMENT

To evaluate the difference in the outcome of this disease as influenced by improved therapy in more recent years, the cases studied have been divided into that group diagnosed and treated before 1950 and that group from 1950 to the present time. These will hereafter be referred to as the "early" and the "late" groups respectively. The immediate treatment in practically every case was bed rest, nothing by mouth, and intravenous fluids (in 64 of the 74 cases). In the early group of 37 patients gastric suction was instituted in only 8 cases, while in the later group, in 20 of the 37 cases suction was started at once. Atropine, or belladonna, every 4 hours was given to only 3 patients in the earlier group and to 8 of those treated after 1950. Banthine was not used before 1950, and since then 16 patients have received it. Antibiotics were given to 11 patients before 1950, and to 16 since that date.

Success in the treatment of patients suffering from pancreatitis demands that an early diagnosis be made. Pancreatitis as a postoperative complication should not be forgotten in which situation an amylase determination on the peritoneal fluid obtained

by paracentesis may point to the correct diagnosis.

Treatment may be considered under four headings: (1) general supportive measures, (2) measures to suppress pancreatic secretion, (3) treatment of the peritonitis, and (4) the question of the role of operation.

General supportive measures. The problem of fluid and electrolyte balance is that of an individual who has suffered a severe injury to a large peritoneal area, with resultant exudation of fluid, intraperitoneal and extraperitoneal, and who, in addition, has suffered further deficit from diminished intake and vomiting. There is definite need for the use of blood and a place for serum albumin. Careful observation of the volume output and the specific gravity of the urine, plus frequent determinations of hematocrit and serum chemistries will serve as guides to replacement therapy. Berk, in discussing the administration of intravenous glucose, cautioned against its use without knowledge of the patient's diabetic status since a transient hyperglycemia or even frank diabetes mellitus may complicate the disease. Five to 10 units of insulin may be given with the glucose, but the potent vagal stimulus of hypoglycemia must be studiously avoided. Potassium and calcium are administered as indicated by serum determinations and clinical evaluation (Chvostek's sign). The Sulkowitch test of urinary calcium also serves as a rough guide for the patient's calcium needs. It is important that effective relief of pain be achieved, preferably with demerol, because it causes minimal spasm of the sphincter of Oddi. In the present series procaine blocks, splanchnic or epidural, were not employed. Antibiotics should be used routinely as a prophylactic measure and certainly, as indicated, to combat definite signs of sepsis.

Measures to suppress pancreatic secretion. In addition to its beneficial effect on the associated paralytic ileus, the use of gastric suction, since it limits the passage of acid gastric contents into the duodenum, retards the hormonal component of the stimuli to pancreatic secretion. Autonomic drugs, such as banthine, block to a degree the neural component of the stimulation and aid in this manner. It is important that adequacy of dosage be determined by a therapeutic effect rather than by standard recommendation.

The treatment of the peritonitis. The effects of peritonitis, chemical and perhaps bacterial, probably constitute the lethal mechanisms of pancreatitis, and measures intended to combat this condition are important. These consist of the use of the semi-Fowler's position, antibiotics, and suction decompression for the paralytic ileus.

The question of the role of operation. Operative procedures were done during the active phase of the disease in 10 patients of the early group, and in 6 of those since 1950. The procedures carried out consisted of:

	Early group No. of cases	*Late group No. of cases*
Drainage	1	2
Cholecystectomy	5	3
Cholecystostomy	0	2
Choledochostomy	0	2
Exploration only	4	0

Whereas nonoperative management is probably preferable in most cases of pancreatitis, there are instances of the hemorrhagic form of the disease with spreading peritonitis wherein a fatal outcome might be avoided by drainage of the lesser peritoneal bursa. Elman, in 1946, reported a series of 17 cases with proved diagnosis of hemorrhagic necrosis. In some of these, conservative therapy was employed. In others, operations were carried out, some on admission, others later. All but 1 of the patients died. The 1 patient who survived underwent drainage of the lesser omental bursa and cholecystostomy in the presence of spreading pancreatitis and incipient shock. In the acute interstitial form of the disease, he believed that the patient would probably survive regardless of the type of treatment. With hemorrhagic pancreatitis, he thought that signs of spreading peritonitis constituted an indication for opera-

TABLE I.—TABULATED RESULTS FROM 33 OF THE 63 SURVIVORS REGARDING THEIR SYMPTOMATOLOGY UP TO THE PRESENT DATE (MAY 1955)

	No. of cases	*Comments*
EARLY GROUP (1940–1949)		
Replies	14	
Recurrences	4	2 with cholecystectomy; symptom-free since 1 died of unrelated disease 10 yrs. later. Developed diabetes 4 yrs. later 1 symptom-free since cholelithotomy 3 yrs. later
No recurrences	8	1 died of unrelated disease 2 yrs. later 1 had elective cholecystectomy
GI symptoms	2	1 had unexplained vomiting attacks 1 complains of "gas pains"
LATE GROUP (1950–1954)		
Replies	19	
Recurrences	5	1 died 8 mos. later (pancreatitis was not directly responsible)
No recurrences	12	1 died of unrelated disease 1 yr. later 3 had elective cholecystectomy, 6–9 mos. later
GI symptoms	2	1 complains of intolerance to certain foods 1 had episodes of substernal pain and low back pain

tion in the hope that adequate drainage of the lesser sac and perhaps cholecystostomy might save the patient's life, but he cautioned against extensive surgery and stressed skillful supportive therapy. Authors of later date agree with these views.

There will occur instances in which the diagnosis of pancreatitis cannot be made with certainty and in which exploratory laparotomy will therefore be a necessity to exclude other lesions. Drainage of lesser omental bursa is probably indicated if acute pancreatitis is found under these circumstances.

In a patient with pancreatitis, the development of progressively increasing jaundice or a distended gallbladder constitutes indications for surgical decompression of the biliary tract.

Alterations in the clinical course of the disease which suggest the development of suppurative intraperitoneal complications of pancreatitis may arise, and under these conditions surgical drainage is considered. Somewhat later, the formation of pseudocysts may occur, the treatment of which is operative.

The management of the recurrent form of pancreatitis is a separate subject, but it is believed that the occurrence of one attack is sufficient indication, after subsidence, for radiologic study of the gallbladder. If malfunction or cholelithiasis is found, surgical management is considered.

OUTCOME, IMMEDIATE AND LATE

Seven patients before 1950 and 4 since that date had a fulminating course ending fatally. In all of these the condition present was acute hemorrhagic pancreatitis except for 1 case of acute suppurative pancreatitis and 1 other, a fatal case of acute interstitial pancreatitis complicated by diabetes. In the 28 cases of acute interstitial pancreatitis and 2 cases of acute hemorrhagic pancreatitis treated before 1950 recovery was uneventful except in 1 case of pneumonia. Since 1950, 34 patients with acute interstitial pancreatitis have recovered. One developed an abscess and another developed a pseudocyst and sinus which was still draining after 5 months so that additional operative treatment was necessary. Four patients in the late group had psychotic episodes of transient nature thought to be of toxic etiology, probably related to marked alcoholism. Three patients had diarrhea during their recovery periods, but in none was steatorrhea proved.

In an effort to obtain follow-up data to the present date on the 63 survivors from this survey group, letters of inquiry were sent to each of them. Thirty-three replies were received from patients and their relatives. The results are tabulated in Table I in summary form. One patient of the early group died 10 years after his initial attack, with respiratory tract complications after an operation for gastrointestinal bleeding. It is of interest that this patient underwent elective cholecystectomy 3 months after his first admission and developed diabetes 4

years later. The death listed under "Recurrences" in the 1950 to 1954 group was a chronic alcoholic readmitted with pneumonia 8 months after her first admission. Autopsy disclosed, in addition to the pneumonia, chronic pancreatitis with fat necrosis, obstruction of the main pancreatic duct, organized thrombi in the pancreatic veins, and duodenal hematoma as well as chronic cholecystitis and lithiasis, periportal fibrosis, and fatty change of the liver.

Using follow-up data from the charts as well as the letters of inquiry mentioned, it was found that 10 patients had some type of biliary surgery as a part of the treatment for their acute disease. Months to years following the acute attack, 12 patients were subjected to cholecystectomy, most of which were elective on the basis of radiologic findings or previous cholecystostomy. In 2 patients sphincterotomy was performed. One of these had a cholecystectomy and common duct exploration at the same time. One patient later developed signs of a common duct stone which required removal. Thus it is seen that at least one-third of the entire group of patients had concomitant biliary disease.

DISCUSSION

The exact etiology of pancreatitis is not known, but evidence suggests an association with primary or secondary disease of the intrahepatic or extrahepatic biliary system. References to this relationship are numerous.

Our patients with acute hemorrhagic necrosis have presented severe clinical pictures when first seen, the patients being semicomatose or in definite shock, and have died within a matter of hours with the exception of 2 patients cited in the following.

CASE 1. A. I., a white male in the 20 to 30 year age group, with a past history only of chronic alcoholism, was admitted in 1941 with epigastric pain radiating through to the back, to the left shoulder, and to the left costovertebral angle, persistent nausea, and vomiting. Physical examination revealed a temperature elevation, abdominal and rectal tenderness, rebound tenderness, a board-like abdomen, and ileus. The white blood count was elevated. Gastric suction and intravenous fluids were instituted and an emergency operation was done without benefit of serum amylase determination. Considerable wine-colored fluid was found in the abdomen, especially in the lesser sac, and the pancreas was found to be swollen. Drains were placed in the lesser sac, and the abdomen was closed. The patient had a febrile course and much drainage, but a progressive and uneventful recovery occurred.

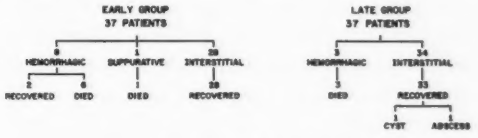

FIG. 3. Outcome in the 74 cases.

CASE 2. J. R. was a white male admitted in 1946. He was in the 50 to 60 year age group and had no previous history of gastrointestinal symptoms or alcoholism. His white blood count was 19,000. He complained of epigastric and right upper quadrant pain accompanied by nausea and vomiting. Physical examination revealed abdominal tenderness, muscle guarding, and ileus. Exploratory laparotomy disclosed bloody fluid and a greatly swollen pancreas, and the abdomen was closed without benefit of drainage. A postoperative determination of the serum amylase revealed 650 units which fell to 426 units during the next 2 days and the patient made an uneventful recovery.

These 2 cases are certainly exceptions to the rule.

There was 1 patient who died with acute interstitial pancreatitis.

CASE 3. P. R., a white male in the 60 to 70 year age group was admitted in 1951, with a history of epigastric pain recurrent since 1945. In the outpatient clinic a gastrointestinal series had been negative and his gallbladder was reported as nonfunctioning. On admission to the emergency room he was cyanotic and had a blood pressure of 80/50, abdominal tenderness and ileus. His serum amylase was reported as 206 units and fasting blood sugar was 249 milligrams per cent. His white blood count was 17,600 and rose to 20,900. He was given atropine, intravenous fluids, and antibiotics. He later developed spasm and rebound tenderness as well as tenderness on rectal examination, and an exploratory laparotomy was done. This revealed an edematous pancreas plus chronic cholecystitis and cholelithiasis, and a cholecystostomy was done. However, the diabetes was resistant to treatment, the blood sugar rose steadily, and the patient succumbed.

Diabetes may present as a serious complication with pancreatitis and, when severe, can make the prognosis for a patient with

interstitial pancreatitis as poor as that for the hemorrhagic form of the disease. Diabetes can also mask an attack of acute hemorrhagic necrosis, as exemplified by 1 of the patients seen, who was brought into the emergency room in diabetic acidosis which was peculiarly resistant to therapy. Death followed, and only at necropsy was a hemorrhagic and necrotic pancreas discovered.

SUMMARY

1. The history, clinical picture, and laboratory findings presented by 74 proved cases of acute pancreatitis are reviewed.

2. The therapy, medical and surgical, is discussed followed by a report of the immediate outcome in these 74 patients. There were 11 deaths.

3. Three case histories, representing atypical courses of the disease, are reported.

4. Late follow-up data indicate that approximately one-third of those patients contacted had at least 1 additional attack of pancreatitis.

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THE MAIN ARTERIES OF THE LARGE INTESTINE

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IN A RECENT PAPER (1) the marginal anastomoses of the arteries to the large intestine were described in a series of 71 cadavers whose arteries had been injected with latex rubber. No description was given of the disposition of the main arteries because this was considered to be adequately described by other investigators using other techniques. However, new circumstances have suggested the desirability of publishing more details of our findings. It was soon apparent that a repetition of purely statistical and conventional analyses would add no important new knowledge. On the other hand, a reproduction of a number of the original maps that we had made would allow surgeons everywhere to share our experience. The individual reader with special interests can derive the statistical or other data that he may require for himself quite easily.

The accompanying drawings illustrate semischematically: (1) the arrangement of all the colic branches of the superior and inferior mesenteric arteries, and (2) the relative sizes of all their anastomoses. A fine line indicates a poor to fair anastomosis, while a thick line indicates a good to excellent anastomosis at the weakest point of each particular loop. These drawings are of the first 45 dissections that were complete throughout. The remainder are not illustrated for convenience and economy.

A quick survey of the drawings shows a great variability of arrangement. As stated earlier, the anastomosis is generally good or excellent in most areas with the exception of the rectosigmoid area where it is often absent. The anastomosis at the left colic flexure is noteworthy for its excellence in most subjects, in spite of the current teaching to the contrary.

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The *superior mesenteric artery* usually (i.e., in three-fifths of cases) has two direct colic branches, much less often (i.e., in one-third of cases) three, and uncommonly four. This is in general agreement with the findings of Helene Pikkieff and of B. W. Ogneff, whose papers, being in German, seem to have been overlooked by English-speaking surgeons. Consideration of our drawings may at first suggest the futility of naming ileocolic, right colic, and middle colic arteries. However, these are useful names if we use them in the following way:

The ileocolic artery is a large artery coming usually from the last branch on the right aspect of the superior mesenteric artery or, less commonly, coming direct from the superior mesenteric. It runs toward the ileocolic region where it breaks up into two or more branches with a variety of anastomotic communications.

The right colic artery is most often a large branch of a common stem artery—common to this and either the ileocolic or the middle colic artery. Much less often, it is a direct branch of the superior mesenteric artery. It runs more or less horizontally toward the ascending colon and usually sends anastomotic branches upward and downward along the colon.

The middle colic artery usually springs from the common stem mentioned (but, occasionally, direct from the superior mesenteric artery) and runs toward the right one-third of the transverse colon where it usually forms anastomotic loops.

In addition, we can give appropriate names to accessory arteries as they occur, e.g., the accessory middle colic artery, being the commonest type (drawings numbered 1, 10, 11, 13, 15, 32, 39, 41, 44, 45).

The *inferior mesenteric artery* always terminates by crossing the brim of the true

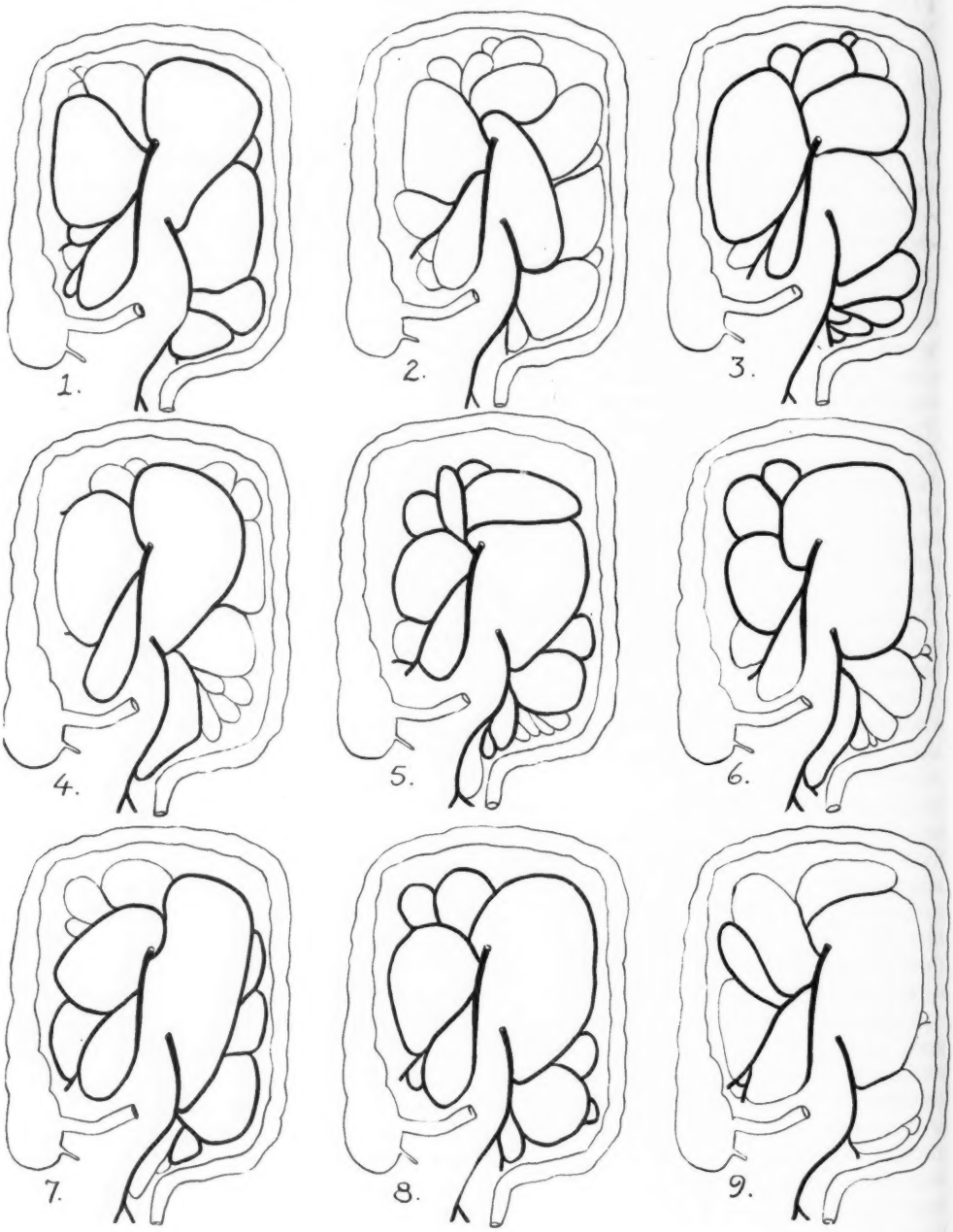
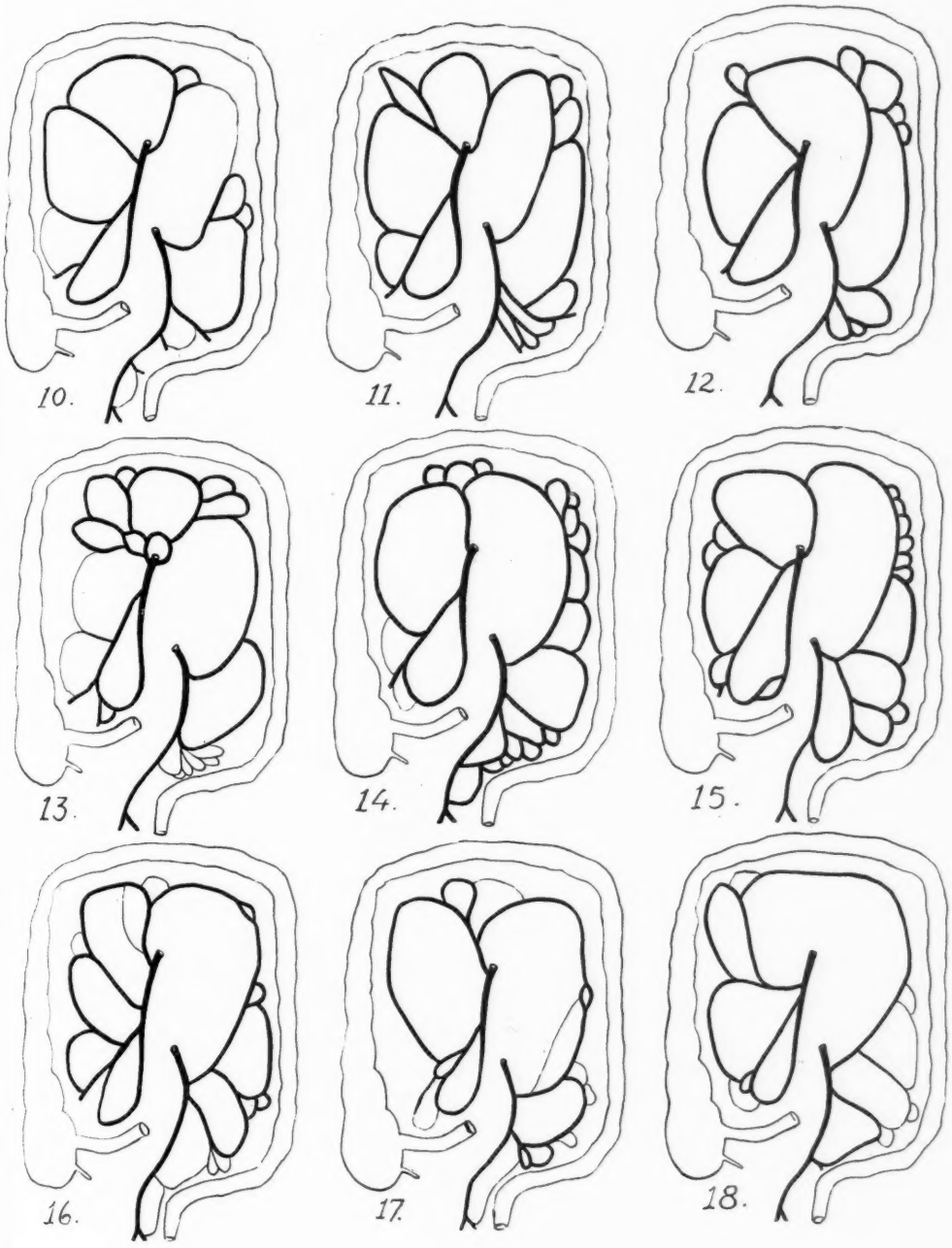


PLATE I.



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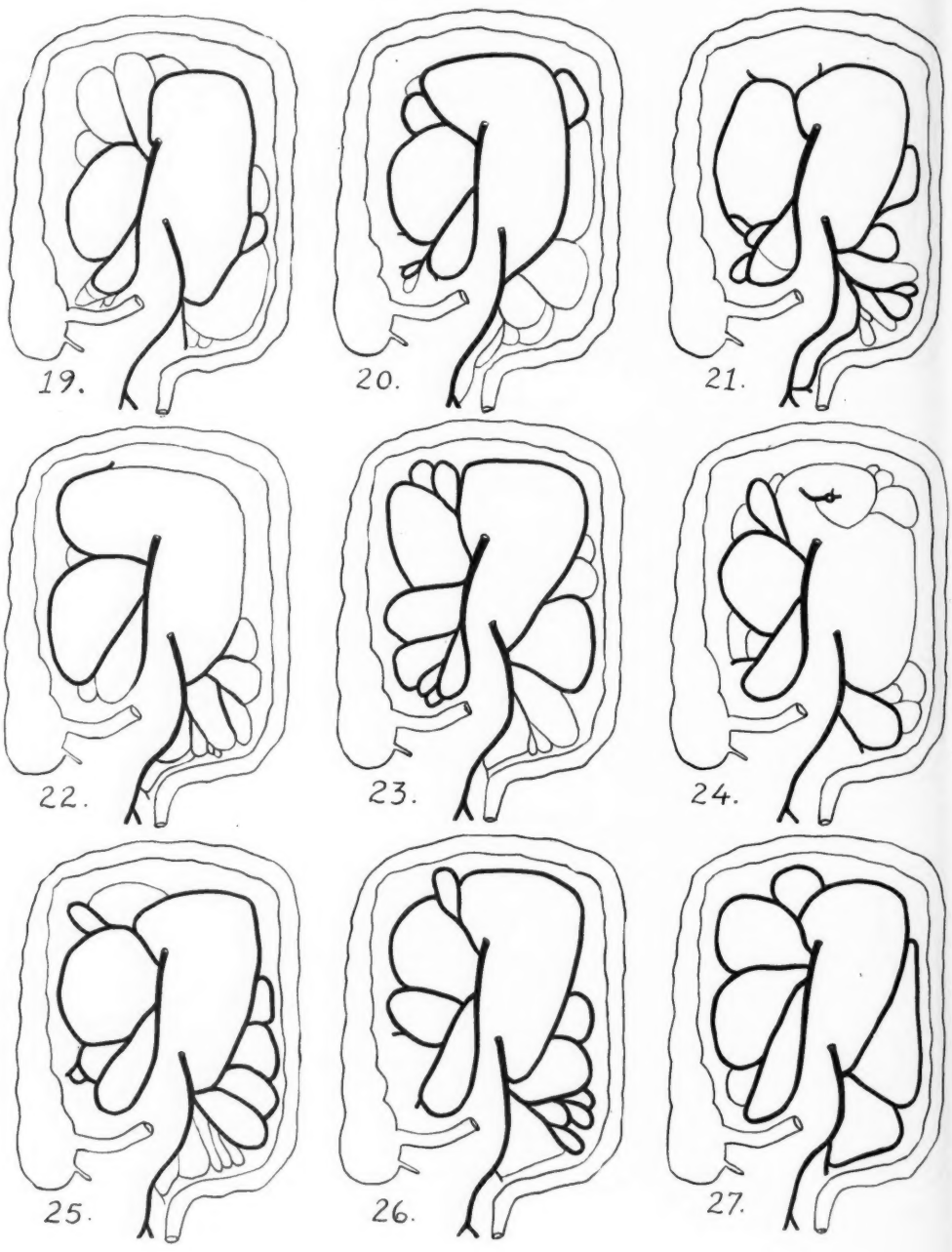

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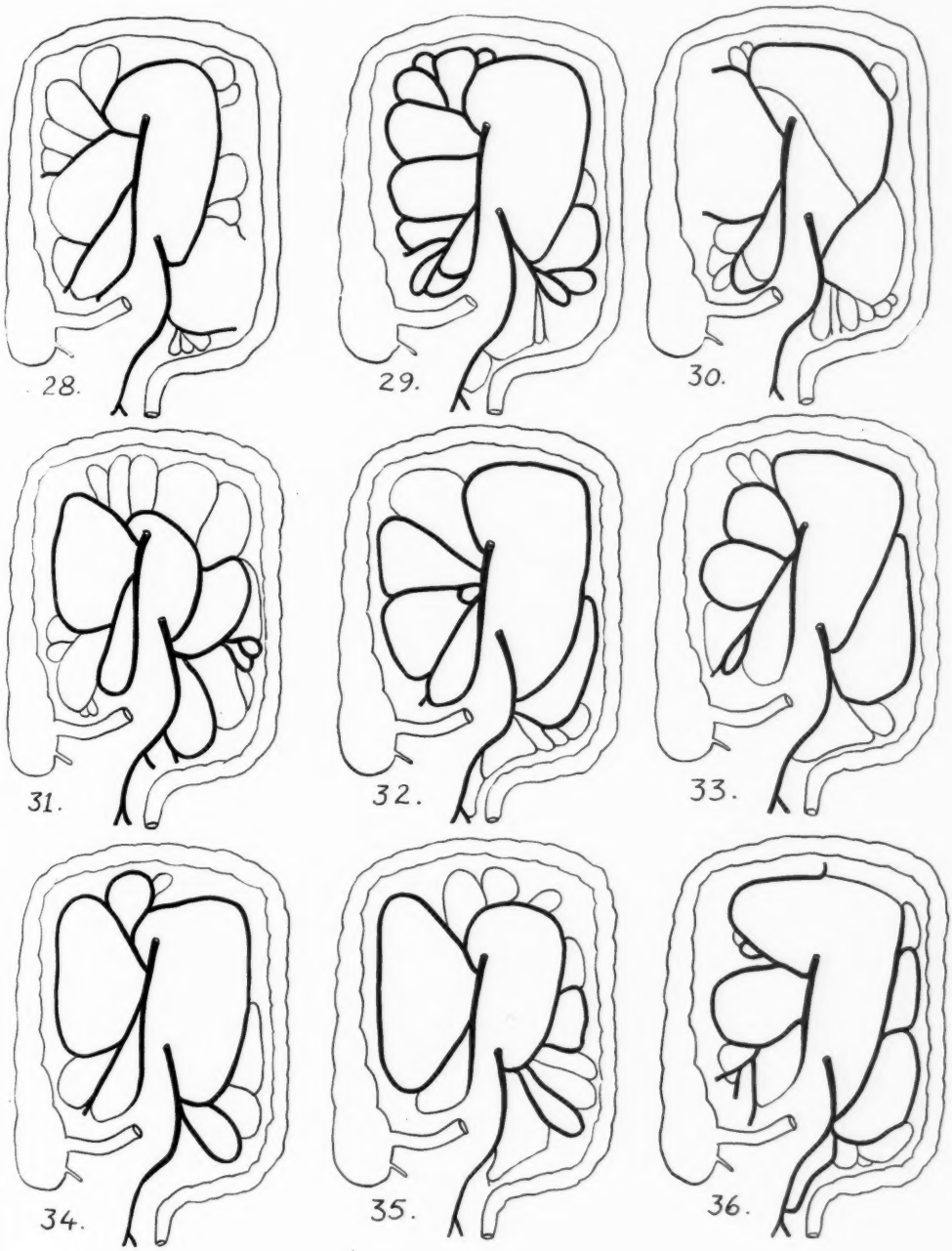

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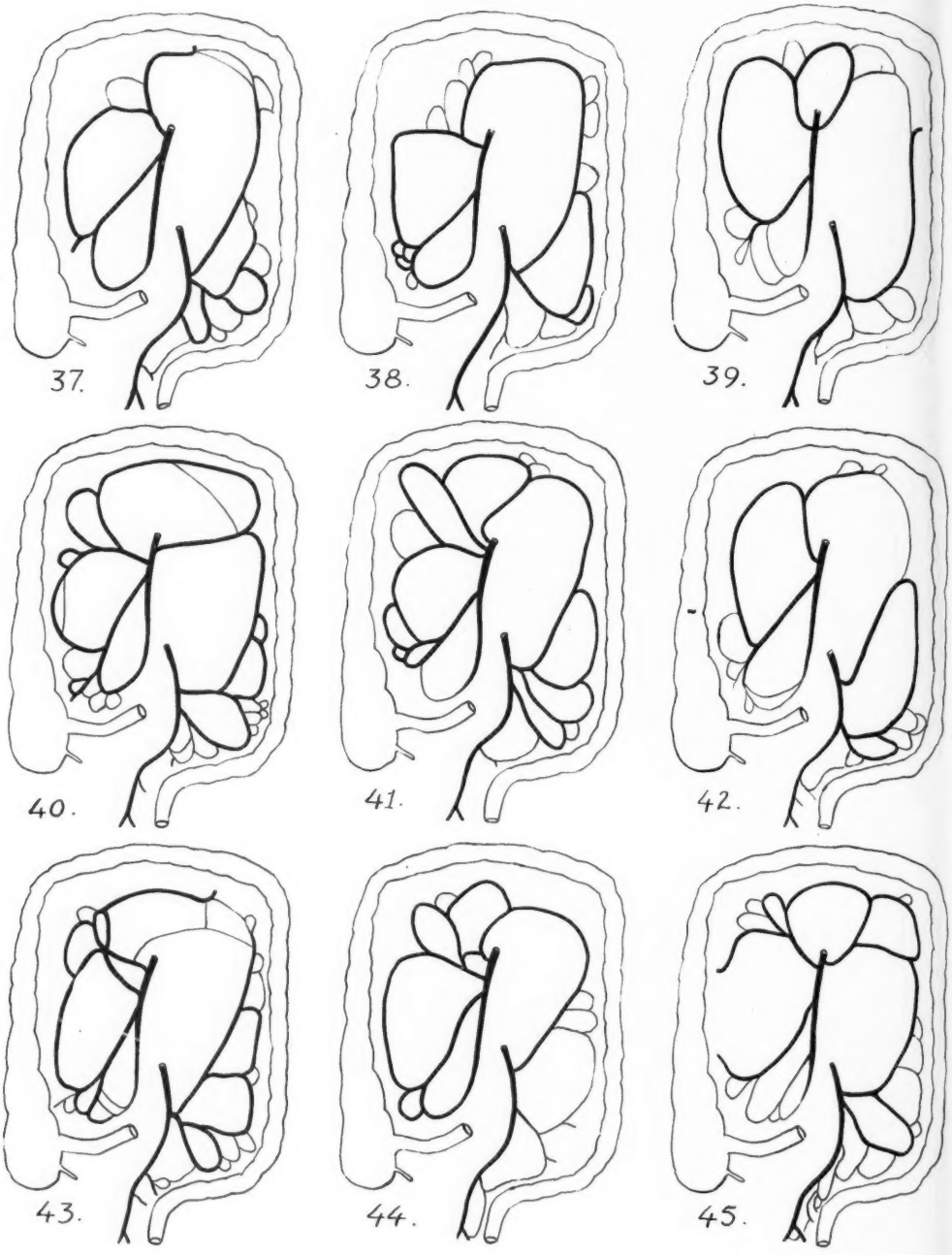

PLATE V.

pelvis where it changes its name to superior rectal artery. The latter passes downward behind the rectum and soon bifurcates. Before crossing the brim of the true pelvis, the inferior mesenteric artery gives off several colic branches to the descending colon and pelvic (sigmoid) colon. Most frequently (in almost one-half of cases) there are only two main branches. In one-third of cases there are three branches and in about one-fourth of cases there are four branches. This is an easy mnemonic to remember: 2:½; 3:⅓; 4:¼. No doubt there will be the occasional subject with only one or with more than four branches. This has been reported by Rubesch.

An interesting anomaly is the *arc of Riolan*. This term should be reserved for a short loop running retroperitoneally from the root of the superior mesenteric artery or one of its primary branches to the inferior mesenteric artery or one of its primary branches. Such a loop is seen in four of the drawings (numbered 2, 3, 30, and 31). Unfortunately, some persons use the term arc of Riolan loosely for the marginal anastomosis at the left colic flexure. This usage can only lead to confusion and should be avoided.

The present series also showed a more unusual anomaly. In drawing numbered 24 there is illustrated a small caliber, communicating loop joining the hepatic artery to the marginal arteries at the left colic flexure. Such an anomaly is a curiosity and no doubt other curiosities may be met with in this field. However, they should interest professional anatomists only.

In conclusion, it may be said that the drawings speak for themselves and that one can obtain from them a wide range of statistical and special information if the need arises.

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EXPERIMENTAL REPAIR OF VENTRICULAR SEPTAL DEFECTS UNDER HYPOTHERMIA

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THE FIRST EXPERIMENTS dealing with the repair of ventricular septal defects carried out in our laboratories utilized closed methods. In 1951 an attempt was made to close such defects in dogs by passing mattress sutures through the septum in such a way as to pull the edges of the defect together when the sutures were drawn taut (9). This method seemed to be safe and was effective in considerably reducing the size of the defect, but not in bringing about complete closure. Recently Cooley has used a similar technique in the treatment of human patients with improvement.

In 1952, experiments which were quite successful in animals were initiated. Using a motor-driven rotary valvulotome it was possible to create ventricular septal defects which remained patent (3). The method of repair entailed the passage of a tube made of autogenous pericardium or latex through the septum and the midportion of the defect so that the defect was occluded when the tube was distended with blood or saline solution. The tube was drawn through by means of a suture attached to a long needle which could be placed accurately within the septum by using a finger in the right ventricular cavity for guidance. Dogs with experimentally produced defects, even those very ill with cardiac failure, tolerated the insertion of the tubes well and secure repairs were accomplished (4). It was a great disappointment that this method had to be abandoned for clinical usage. Initial trial in very ill patients lent some encouragement (8). However, after treating 8 patients by this method we discontinued its use because, though we could ordinarily close the defect completely or nearly so, the mortality was prohibitively high. Furthermore, follow-up studies in dogs revealed the fact that the latex tubes, which were easier to insert than the pericardial tubes, sometimes underwent late rupture with recurrence of the defect.

We, therefore, directed our attention to open methods. The present study reports our experiences with the repair of defects under hypothermia. Recent studies concerning the protective value of procaine blockade of the right atrial wall and better methods for dealing with coronary air embolism and ventricular fibrillation gave us hope that repair under hypothermia might be a feasible procedure (5–7).

From the Department of Surgery, Indiana University School of Medicine, Indianapolis, Indiana. Aided by grants from the Indiana Heart Foundation and the James Whitcomb Riley Memorial Association, and by a contract between the Office of Naval Research, The United States Navy and Indiana University.

MATERIALS AND METHODS

Healthy mongrel dogs were used. They were anesthetized with 3 to 4 cubic centimeters of 5 per cent thiopental sodium injected intravenously. Tracheal intubation was then carried out and, until the operation was over and the animal rewarmed, hyperventilation with oxygen was maintained by mechanical insufflation of the lungs at the rate of 35 to 45 times per minute. The dogs were cooled to a temperature of 30 degrees C. by emersion in a bath of ice and water.

The operations were carried out aseptically through a right fourth or fifth intercostal space incision. The venae cavae were dissected free, the azygos vein was ligated, and the pericardium opened. The right atrial

TABLE I.—EXPERIMENTS WITH REPAIR OF VENTRICULAR SEPTAL DEFECTS MADE SEVERAL MONTHS BEFOREHAND

No. of dog	Temp. in degrees C.	Period of caval occlusion in minutes	Result
30	27	8	Uneventful—sacrificed 21 days later
35	26	8	Uneventful—sacrificed 15 days later
76	26.8	6.5	Uneventful—sacrificed 22 days later
79	27	7.5	Death from untreated cardiac standstill
85	26	5	Uneventful—sacrificed 28 days later
86	27	8	Uneventful—sacrificed 15 days later
109	27	8	Death from untreated coronary air embolism and ventricular fibrillation
122	27	4.5	Uneventful—sacrificed 23 days later
139	27	7	Death from untreated cardiac standstill in rewarming period
145	27	7	Death from untreated cardiac standstill. Tricuspid insufficiency
147	26	7	Death from untreated ventricular fibrillation
150	27	7	Death from untreated coronary air embolism and ventricular fibrillation
153	27	6	Death from untreated ventricular fibrillation
165	26.8	8.5	Uneventful—sacrificed 15 days later
168	27.2	7	Uneventful—sacrificed 19 days later
179	27	5	Death from untreated ventricular fibrillation

wall and the atrial-superior vena caval junction were infiltrated with procaine as previously described (7). By the time this had been accomplished the temperature of the dog had usually fallen to 26 or 27 degrees C. The venae cavae were occluded and the right ventricle was opened widely through an incision of from 4 to 7 centimeters in length.

There were two groups of experiments. In the first a high ventricular septal defect had been made, by the method previously reported(3), from 2 to 6 months beforehand. In these animals as soon as the ventricle was opened and its cavity emptied of blood by aspiration, the defect was located and occluded momentarily with the tip of a finger. The defects could ordinarily be closed with 2 or 3 interrupted sutures of silk.

In the second group of animals a high ventricular septal defect was made and closed at the same operation. The exposed ventricular septum was grasped with a toothed forceps and a defect 6 or 8 millimeters in diameter created with a fine scissors. The defect was dilated somewhat by spreading the blades of a Kelly clamp. It was then temporarily closed by placing a finger over it. Repair was then accomplished by approximating the edges with from 2 to 4 silk sutures.

In both groups of animals the incision in the ventricular wall was rapidly sutured with continuous silk after the ventricle was filled with saline solution. The superior vena caval clamp was then released; the inferior vena caval clamp was removed from 2 to 3 minutes later. Once the chest wall was repaired, the surviving animals were rewarmed in a bath of water kept at 45 degrees C. They were sacrificed from 1 to 170 days later. At the time of sacrifice the heart was examined carefully. Electrocardiographic studies were made during the procedure and at intervals thereafter.

RESULTS

There were 16 animals in the first group. High septal defects 6 millimeters in diameter had originally been produced in 30 dogs. Three animals had died during the operation of ventricular fibrillation and 1 later the same day from septal ischemia due to excision of a portion of the septal artery in making the defect. Nine died of heart failure in from 1 to 8 days after operation. One was killed in a fight (Fig. 1). In the 16 animals reoperated upon under hypothermia (Table I) considerable difficulty was encountered because of adhesions between the heart and pericardium. After the pericardium was dissected free, the landmarks of the heart were not clear. It was difficult and sometimes impossible to carry out an effective procaine injection. In 3 instances attempted injection was not followed by slowing of the pulse nor

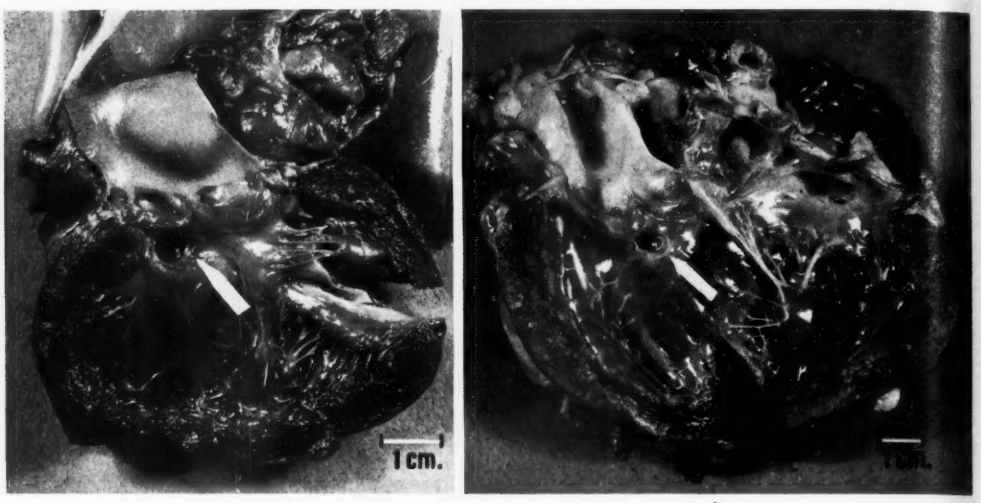

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FIG. 1. Ventricular septal defects. a, Infant's heart, b, Experimentally produced defect in dog's heart. The similarity in location is evident.

changes in the *P* wave. Eight of the 16 dogs survived successful repair of the defect while 8 died during the operation or the rewarming period. In general, those with the fewest pericardial adhesions survived the procedure, while those with the most troublesome adhesions died. Two animals died of coronary air embolism and fibrillation. The 3 animals with ineffectual attempts at procaine blockade developed ventricular fibrillation and died. Cardiac standstill occurred in 3. No attempt was made to carry out resuscitation in these 8 animals. The 8 dogs which survived the repair were sacrificed in from 15 to 75 days. The defect was closed in all.

In the second group of 16 animals, the injection of the atrial wall was successfully accomplished as evidenced by slowing of the pulse and disappearance or alteration of the *P* waves. The defects were produced with ease. The closure of the defects, however, was more difficult than in the first group. Sutures passed through the septal muscle close to the defect tended to pull out, and fairly large bites of tissue had to be included in the sutures. Furthermore, it was much more difficult to keep the defect closed by finger pressure during its repair than was true in the group with old well healed defects. The incidence of air embolism was high (Table II). Some air was seen in the coronary arteries in 8 animals. In 4 there was massive embolism with fibrillation. All were successfully defibrillated with 1/10 second 170 volt electric shock after the removal of the air by vigorous cardiac massage during cross clamping of the ascending aorta. Fibrillation did not occur in the 4 animals with a smaller amount of air in the right coronary artery. In them the air was quickly removed by milking it out from the incised ends of 1 or 2 of the small terminal coronary branches. The hearts which were becoming progressively cyanotic regained normal pink color as soon as the air was removed and cardiac contractions became stronger. In only 1 animal did ventricular fibrillation develop prior to the occurrence of a gross coronary air embolism. In this exceptional case, in order to prevent coronary air embolism a Satinsky clamp had been placed across the base of the heart so as to occlude the proximal portion of the aorta. Thus ventricu-

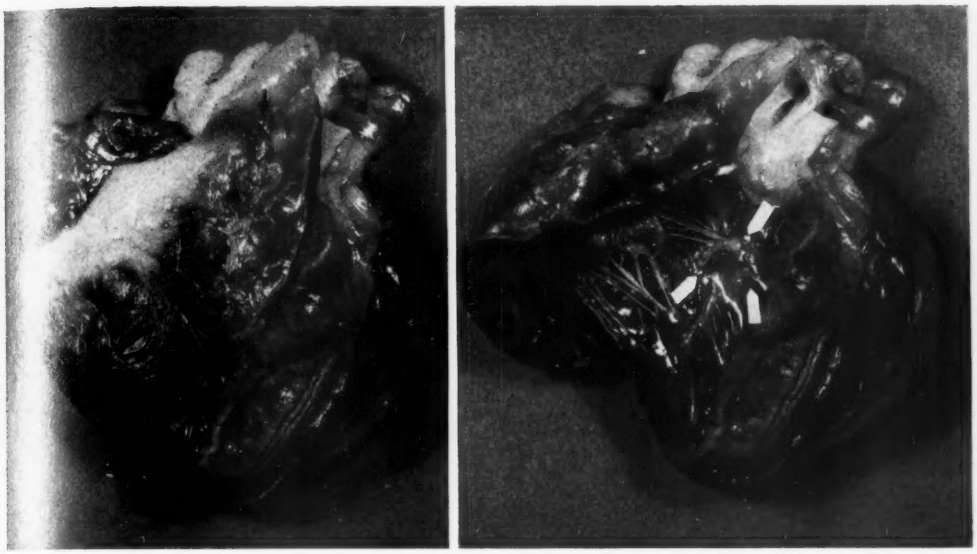

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FIG. 2. Postmortem specimen of dog 743, 25 days after repair of ventricular septal defect. The ventriculotomy incision is well healed and the septal defect securely closed.

lar fibrillation developed altogether in 5 animals, 4 of which had gross air embolism. Four additional dogs with coronary air embolism did not develop fibrillation and no difficulty whatever was encountered in the remaining 7. All survived the effective suture closure of the defect and were sacrificed in from 1 to 170 days. Postmortem examination revealed the defects to be closed (Fig. 2). In 2 animals a mural thrombus was present.

In most of the animals the ventricular complex was inverted in the electrocardiographic tracing immediately after the closure of the defect (Fig. 3). In a few permanent electrocardiographic changes ensued, but in most of them the electrocardiographic changes returned to normal some time afterward.

DISCUSSION

When experimentally produced defects are repaired a long while after their production certain difficulties arise because of adhesions between the heart and pericardium. Taufic and Lewis lost 7 of 12 animals when repair under hypothermia was attempted at a second operation. We had a 50 per cent mortality in a group of 16 animals in which repair was carried out some months after the production of the defect. To be sure, the mortality might have been considerably less had we made any effort to treat the air embolism, fibrillation, and standstill which accounted for these deaths. In our hands a defect can be made and closed under hypothermia without mortality with the use of procaine blockade of the superior vena caval atrial junction and vigorous treatment of coronary air embolism and ventricular fibrillation when it occurs.

Using hypothermia, Taufic and Lewis made, and closed by suture, ventricular septal defects in 9 dogs. Four died as the result of operation and ventricular fibrillation was noted in 7, though it was thought responsible for the death of only 1. When they made ventricular defects in the same way and repaired them by applying a plastic plate, the results were better. Only 2 of 12 animals died

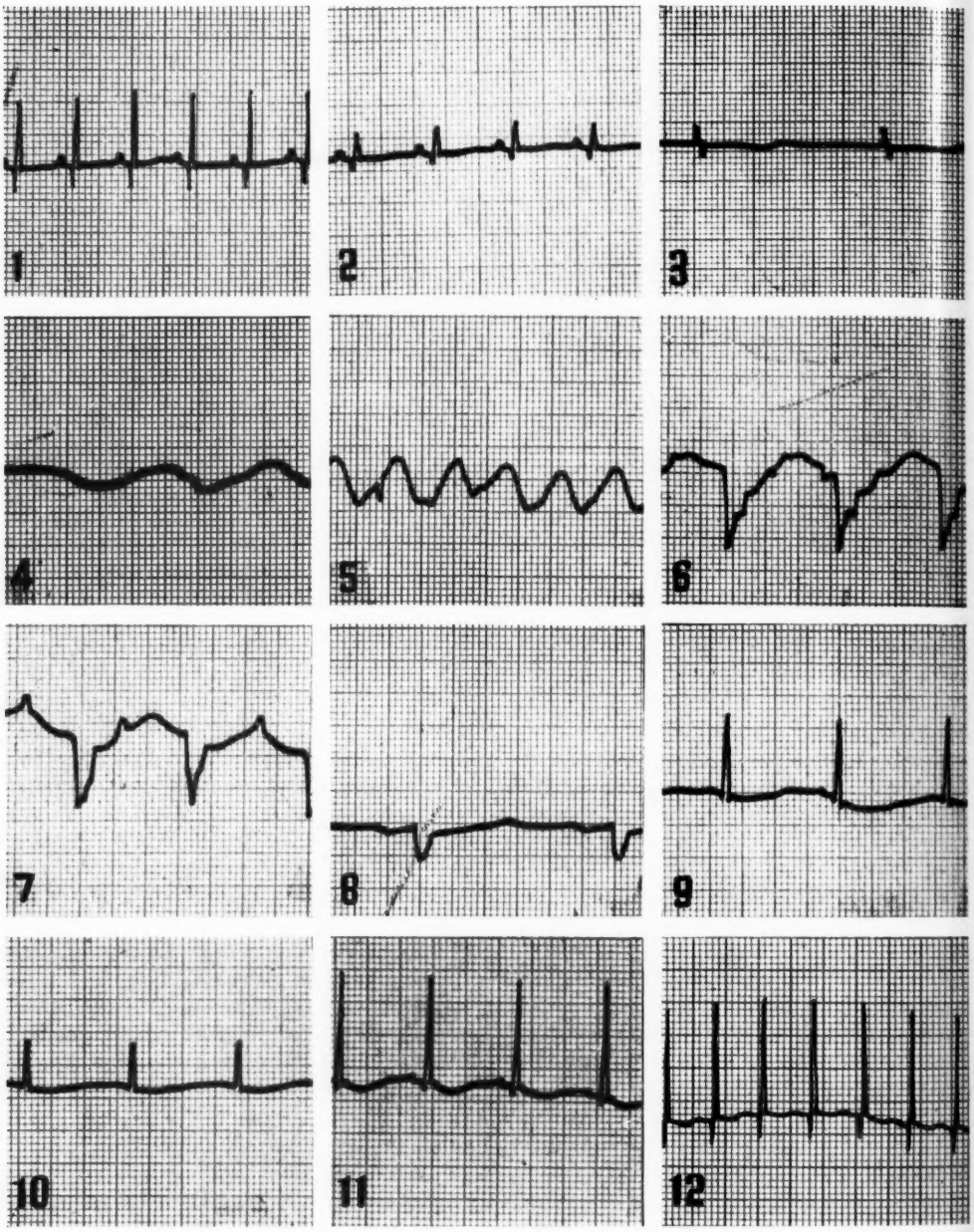

FIG. 3. Electrocardiographic tracings (lead *2*) of dog 752. 1, At 33 degrees C. 2, At 27 degrees C. before procaine injection. 3, After injection. 4, Fibrillation due to air embolism. 5, Fibrillation after cardiac massage and intracardiac epinephrine. 6, Immediately after defibrillation. 7, After closure of pericardium. 8, After closure of chest. 9, At 31 degrees C. 10, At 33 degrees C. 11, At 36 degrees C. 12, Twenty-three days later.

TABLE II.—EXPERIMENTS WITH PRODUCTION AND IMMEDIATE REPAIR OF VENTRICULAR SEPTAL DEFECTS

No. dog	*Temp. in degrees C.*	*Period of caval occlusion in minutes*	*Result*
68	27	6	Uneventful—sacrificed 14 days later
69	26	5.5	Uneventful—sacrificed 30 days later
69	25.5	7	Uneventful—sacrificed 10 days later
69	26.2	6	Resuscitation from air embolism and ventricular fibrillation—sacrificed 27 days later
69	26.8	6	Resuscitation from air embolism without fibrillation—sacrificed 1 day later
69	26	6	Uneventful—sacrificed 39 days later
6	26.2	6	Resuscitation from air embolism without fibrillation—sacrificed 33 days later
7	26.5	8	Resuscitation from air embolism and ventricular fibrillation—sacrificed 170 days later
-	27	7	Resuscitation from air embolism without fibrillation—sacrificed 10 days later
-	26.5	7	Uneventful—sacrificed 10 days later
-	26	8	Resuscitation from air embolism without fibrillation—sacrificed 28 days later
-	26	7	Uneventful—sacrificed 2 days later
-	26.5	7	Resuscitation from ventricular fibrillation—sacrificed 25 days later
-	27	5	Resuscitation from air embolism and ventricular fibrillation sacrificed 22 days later
-	26	8	Resuscitation from air embolism and ventricular fibrillation sacrificed 6 days later
6	26	5.5	Uneventful—sacrificed 150 days later

within the first 3 days and an additional dog later. Ventricular fibrillation occurred in 2 and both were resuscitated.

Using an artificial heart-lung apparatus, Gibbon and his associates made and repaired ventricular septal defects in 20 dogs. There were 8 operative deaths and in only 9 of the 12 survivors was closure complete at the time they were sacrificed.

Warden and his group created and closed ventricular septal defects with the aid of controlled cross circulation. In 23 the perfusion was 3 times the calculated azygos flow. There were 15 deaths, and 2 survivors had irreversible brain damage. In 14, perfusion was 3 to 5½ times the calculated azygos flow. There were 4 deaths.

Thus, from the standpoint of experience with the creation and repair of ventricular defects in dogs by open operation, the method we have used yields the lowest mortality. This does not, of course, necessarily mean that it will prove to be best for use in cases of humans. We have successfully closed a ventricular defect by this method in a very ill 11 month old child. Ventriculotomy was performed at 27 degrees C. during a 7½ minute period of complete vena caval occlusion. The infant withstood the procedure well and no thrill or murmur was evident afterward. There was some tachycardia but no definite evidence of congestive failure. About 8 hours after operation she developed some twitching of her upper extremities and she expired 7 hours later. Postmortem study revealed no cause of death. The defect was closed. The question whether death was due to cerebral air embolism could not be answered.

Though the use of hypothermia with the auxillary aids employed in our study may make ventricular exploration in man much safer than has previously been thought possible, it would have two potential disadvantages. For one thing, the time for work within the opened ventricle would be brief and perhaps inadequate in many cases. For another, there would be more danger of air embolism than with some perfusion method which might keep the aortic valve closed. At any rate, the measures used in making it possible to create and repair ventricular septal defects in dogs should contribute to the over-all safety of hypothermia.

SUMMARY

By utilizing certain safeguards and methods for managing complications, ventricular septal defects were created and repaired in dogs without mortality.

Doubt is expressed that this method will prove best for clinical repair of ventricular septal defects.

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CHOLEDOCHODUODENOSTOMY IN THE TREATMENT OF CHOLANGIOHEPATITIS

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CHOLANGIOHEPATITIS is a pyogenic infective condition of the biliary tract that is frequently encountered in South East Asia. It is peculiarly associated with the Chinese race so that it has come to be known domestically on our unit as the Hong Kong syndrome, although that name suggests an undue topicality. Among the indigenous population, its incidence is considerably greater than inflammatory disease of the gallbladder alone. This is in striking contrast to surgical experience in Western countries where acute and chronic cholecystitis holds pride of place among disorders of the biliary tree. The racial predilection of cholangiohepatitis is further emphasized by noting that we have never seen an example in Europeans living in this area, nor do we know of any recorded cases.

The clinical features of cholangiohepatitis are sufficiently constant as to present no difficulty in diagnosis to one acquainted with the condition.

The disease syndrome was first described by Digby (2) and subsequent reports have appeared in the literature from time to time, notably one by Cook, Hou, Ho, and McFadzean in 1954.

As an introduction to the surgical management of cholangiohepatitis, with which this paper is mainly concerned, the salient features of the disease will be reviewed so that the problems posed to treatment may be appreciated.

CLINICAL FEATURES

Clinically the patient presents with a triad of pain, jaundice, and fever to which may be added a palpable gallbladder as a variable finding. Typically the patient is Chinese of either sex in the 20 to 40 age group. A history of previous similar attacks may be obtained.

Pain is of rapid onset, well localized to the right upper quadrant of the abdomen, either continuous or colicky, and often severe. Concomitant pain in the back or shoulder tip is not usually admitted. Jaundice is manifest in the majority of patients but, if subclinical, is indicated in the history by the patient's observation that the urine is "tea-colored." Clay-colored stools are less frequently observed. Confirmation of subclinical jaundice is further obtained by noting positive tests for bile in the urine and an icteric serum bilirubin level. Subjective fever is reflected in a raised temperature reading of the order of 101 to 102 degrees F.

On clinical examination of the abdomen, marked tenderness is found in the area subjacent to the right subcostal margin extending downward to a variable level but not usually below the umbilicus. In many patients, an enlarged gallbladder can be distinctly palpated at the site of maximum tenderness but rigidity of the overlying abdominal muscles may prevent the examiner's hand from appreciating this feature.

PATHOLOGY

Pathologically, a suppurative inflammation due to Bacillus coli exists in association with obstruction to the bile ducts, consequent upon the presence of inspissated biliary debris in the ducts which may consolidate into friable multifaceted calculi. Their distribution is variable. They may be present uniformly throughout the whole duct

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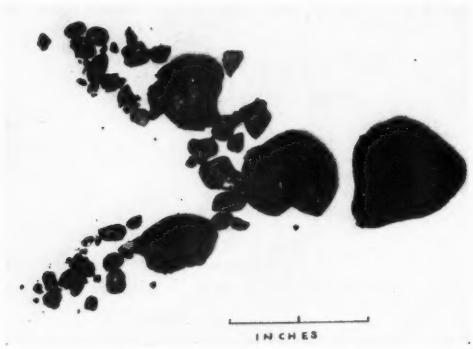

FIG. 1. Calculi evacuated from the bile ducts of 1 patient, arranged as they were found at operation.

system or be localized principally in either the intrahepatic or extrahepatic ducts. It is uncommon to find them concomitantly in the gallbladder which shares little in the pathologic changes involving the ducts. Evidence of obstruction, i.e., jaundice, palpable gallbladder, however, is encountered in some patients without the subsequent disclosure of stones, and in these cases we presume that spasm of the sphincter of Oddi or inflammatory edema of the ampulla of Vater is the causal factor.

What is the pathogenesis of cholangiohepatitis is a question to which we have no ready answer. Instead we can only point to some observations on associated factors and formulate an opinion.

Basically, cholangiohepatitis consists of infection associated with obstruction, and to decide which of these two factors is precedent is fundamental to an appreciation of etiology. If stone formation is the prime mover then superadded infection might reasonably be expected to follow obstruction and stasis as such a circumstance has its counterpart in other situations, for example, salivary calculi and suppurative sialoadenitis, renal stone, and pyelonephritis. This hypothesis still leaves the origin of the biliary calculi unexplained. The stones contain a considerable amount of pigment and, that being so, could be formed as a consequence of a disturbance of bile pigment metabolism in the same way that pigment stones are associated with the hemolytic anemias. There is, however, no suggestion of such a disorder in these patients and a more satisfactory argument can be evinced for regarding infection the forerunner, as follows. Stones are not always found to be present in some patients otherwise displaying all the typical manifestations of the disease as was pointed out earlier but in whom infection of the ducts clearly exists. It would seem then that impaction of stones in the ducts although a frequent concomitant is not an essential factor in the development of cholangiohepatitis, whereas infection is common to all cases. What determines the advent of infection in the bile ducts at the outset? It is most unlikely that infection is primary or else cholangiohepatitis would be a condition universally encountered. Rather must there be some initiating stage making the biliary tree vulnerable to secondary infection. In looking for such a factor there is much to incriminate liver fluke infestation. This notion is attractive in that it would explain the geographic and racial incidence of cholangiohepatitis and be at the same time in accord with the frequent finding at operation of Clonorchis sinensis in bile drained from the ducts. It must be admitted that the observation is not invariable but there is no reason to suppose that a self-limiting infestation had not previously existed in these exceptions as clonorchis usually dies in the ducts after producing ova. Clonorchis sinen-

TABLE I.—SUMMARY OF THE CLINICAL FEATURES OF CHOLANGIOHEPATITIS IN 19 PATIENTS

	No. of patients	*Per cent*
Pain	19	100.
Jaundice	16	84.2
Subjective fever	7	32.1
Dark urine (Patients observation)	11	57.8
Bile in urine (Clinical test)	11	57.8
Clay stools	2	10.5
Palpable gallbladder	10	52.6
Muscle guarding	14	73.6
Previous history	13	68.4
Stones or debris	14	73.6
No stones	5	26.3
Adult clonorchis in bile ducts	6	31.5

sis is a parasitic fluke having the adult stage of its life cycle in human bile ducts, and in those of dogs, cats, pigs, and rats which all abound in Chinese villages.

During the course of development it passes through two intermediate stages, the first in one of several different species of snails and the second in one of an equally large variety of edible fresh water fish. Infestation occurs by eating the fish either raw or parboiled, for the encysted cercaria can resist temperatures of up to 70 degrees C. for 15 minutes. Infestation rate is high (38 per cent) as indicated by routine stool examination for all patients admitted to hospital, while postmortem figures are even higher.

As far as we are aware the only way in which alimentary infestation differs in the local population from that in other tropical countries is in respect of Clonorchis sinensis. We think this finding noteworthy in explaining the distribution of cholangiohepatitis.

Chronic irritation of the bile duct walls could produce a state of lowered tissue resistance to Bacillus coli infection, the organisms reaching the liver via the portal circulation as a portal bacteremia and being excreted in the bile.

Infestation, infection, and obstruction, then, are the suggested sequence leading to the clinical syndrome of cholangiohepatitis, and this hypothesis makes the associated biliary stones secondary to infection and stasis in the ducts.

Spontaneous resolution of an attack commonly occurs, but the respite is only temporary and recrudescence of obstruction and infection is inevitable with the attendant dangers of damage to the liver parenchyma and threat to hepatic function. With the latter in mind treatment of cholangiohepatitis at the earliest opportunity is desirable, and it is to treatment that we will next direct our comments.

TREATMENT

When calculus obstruction of the common bile duct occurs secondary to cholecystolithiasis, treatment is clearly indicated and the results are satisfactory. Cholecystectomy removes the site of calculus formation, and evacuation of the common bile duct effectively deals with the obstruction for all time unless a residual stone is overlooked. It is not so with cholangiohepatitis. Here the ducts themselves are the site of stone formation, and the gallbladder is an innocent bystander and useful safety valve. Evacuation of the ducts on one occasion does nothing to ensure against future reformation of calculi as the circumstances of their original genesis continue unchanged. While the nature of these circumstances remains unknown to us we have to direct our attention to those features of the disease which appear amenable to treatment. At the outset, relief of obstruction and drainage

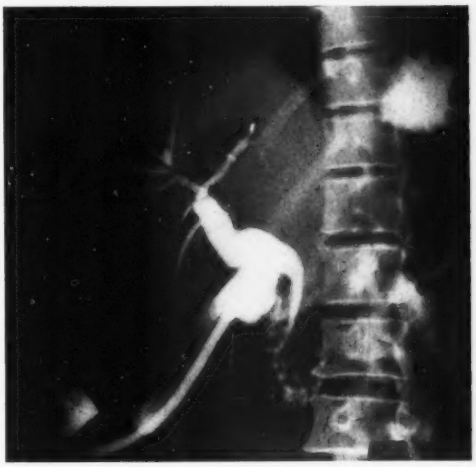

FIG. 2. Cholangiogram of patient treated by T-tube drainage of the common bile duct. The intrahepatic ducts are well outlined by dye, some of which has passed into the stomach and duodenum.

TABLE II.—HELMINTHIC INFESTATION IN 1,013 CONSECUTIVE PATIENTS

Positive findings	*No. of patients*	*Per cent*
Ascaris lumbricoides	225	22.2
Ancylostoma duodenale	111	11.4
Clonorchis sinensis	387	38.2
Fasciolopsis buski	10	1.0
Trichuris trichiura	346	34.2

Showing the incidence of helminthic infestation in 1,013 consecutive patients admitted to the wards of the Professorial Surgical Unit of the Queen Mary Hospital, Hong Kong, during the period 1951–1952, as denoted by the finding of ova in the stools.

of the bile ducts are called for as prerequisite of controlling the infection. We believe that conservative measures have little or nothing to offer these patients unless performed as a part of preoperative preparation when necessary.

The group of patients in whom there are no stones could no doubt be satisfactorily treated medically during an attack by antibiotics, spasmolytic agents, and sedation. Unfortunately, there is no way of distinguishing these cases preoperatively as the calculi associated with cholangiohepatitis are usually nonradio-opaque. It is preferred, therefore, to regard all patients as having calculus obstruction and to accept the diagnosis of cholangiohepatitis as an indication for surgical intervention.

FINDINGS AT OPERATION

On opening the abdomen via a subcostal or upper transrectus incision, the gallbladder is seen to be tensely distended. There are no adhesions in its vicinity and access to the bile ducts is unhindered. A little bile-stained fluid may be found in Morison's pouch. The free border of the gastrohepatic omentum is swollen with inflammatory edema giving it a gelatinous appearance and cloaking its contained structures. The extrahepatic ducts are markedly dilated, the dimensions of the common duct at times approximating those of the small bowel. Palpation of the ducts reveals the presence of calculi in the majority of cases and it is not unusual for the whole of the accessible duct system to feel "solid" with the impacted stones. After incising the peritoneum over the common bile duct, the latter is identified, then opened between stay sutures. Thick purulent bile, often containing several adult clonorchis, and occasionally an ascaris worm, gushes out and is sucked away. On decompressing the duct the gallbladder can now be emptied by gentle pressure. When no stones are encountered, a bougie passed distally into the common bile duct slips easily into the duodenum, a finding that makes explanation of the obstruction in such cases a matter of conjecture. The more usual finding of organic obstruction occurs either because of large irregular friable calculi or as inspissated biliary mud. Evacuation of the former is best achieved with stone forceps and the latter by using a malleable scoop. Individual stones may be of such a size that it is necessary to reduce them to smaller pieces in situ before being able to withdraw them through the choledochostomy opening. Thorough exploration of the bile ducts is required from the ampulla of Vater to as far up the hepatic ducts as instrumentation will allow, as stones may be present in any part of the system. The extent of calculus formation in cholangiohepatitis can be gauged from Figure 1 which illustrates the stones removed from 1 patient and arranged as they were found at operation. When clearance of the ducts is complete as far as possible and the patency of the ampulla of Vater ensured by the unhindered passage of a sound into the duodenum, irrigation with saline is carried out to remove any residual particles of debris.

Up to the end of 1953 it was our custom to complete the operation at this stage by inserting a T tube into the common bile duct with three objects in view: firstly, to provide for free drainage of infected bile during the immediate period of postoperative convalescence; secondly, in order to carry out cholangiography by outlining the biliary tree with radio-opaque fluid, such as diodrast, introduced through the T tube. By this means any residual stones could be demonstrated as radiologic filling defects (Fig. 2) and the effectiveness or otherwise of the surgical toilet of the ducts assessed. In a high proportion of cases, stones were disclosed remaining in the intrahepatic ducts. When situated high up in the ducts such stones were inaccessible to instrumental removal and provided a major problem in the management of these patients. The presence of residual intrahepatic stones constituted the third reason for inserting a T tube as a means of irrigating the bile ducts in an effort

FIG. 3. Barium meal roentgenogram showing reflux of barium into the bile ducts through the choledochoduodenostomy stoma.

FIG. 4. Roentgenogram taken half an hour after the film of Figure 3 with the patient erect and demonstrating the rapidity of drainage from the hepatic ducts.

to complete the clearance of calculi. Substances that we used at various times for this purpose included sterile saline for a mechanical flushing action, ether in an attempt to dissolve the stones, and streptokinase and streptodornase for their fibrinolytic action on the binding matrix of the calculi, in the hope that the disintegrated stone would pass down the ducts and be evacuated. Irrigation of the ducts with these substances was carried out daily during the patient's stay in hospital and subsequently twice weekly in the outpatient clinic with periodic cholangiograms to check progress. Following initial tube drainage for the first 10 days after operation, the T tube was clipped off to restore flow into the duodenum and the patient discharged from hospital with the T tube still in position for outpatient treatment which in many cases lasted from 3 to 6 months. The results of this regime on the whole were disappointing in effecting clearance of the intrahepatic ducts as assessed by cholangiography and, we thought, did not justify the discomfiture to the patients of maintaining a T tube in situ for a protracted length of time. Again, ether and fibrinolytic agents were found to have no advantage over saline for duct irrigation. With ether, patients would experience pain as the liquid vaporized and distended the bile ducts. An initial encouraging result with streptokinase was not supported by later experience and had the disadvantage of being expensive.

Another shortcoming of the T-tube drainage policy was that it did not mitigate the possibility of recurrence of cholangiohepatitis after the tube was withdrawn.

At the time that we became conscious of these shortcomings of choledochostomy drainage for cholangiohepatitis, a procedure as practiced by the late Professor Digby in this colony was brought to our notice by G. H. Thomas, who himself was the first to operate on a patient with cholangiohepatitis in Hong Kong in 1912. Digby (2, 3) recorded in the literature the cases of 4 patients on whom he had carried out

FIG. 5. Barium meal roentgenogram taken with the patient in the erect posture and disclosing the presence of gas in the biliary tree.

choledochoduodenostomy. Briefly, his management consisted of a first stage laparotomy and cholecystostomy followed by an elective choledochoduodenostomy at a second stage performed 3 to 6 weeks later.

Since January 1954, we have adopted this policy with certain modifications and during that year treated 19 patients. We deviate from Digby in preferring to carry out choledochoduodenostomy at the initial exploratory operation and a side-to-side anastomosis instead of an end-to-side union which he advocated.

OPERATION

Preoperatively a Ryle's tube is passed and the stomach is aspirated, an intravenous infusion is set up, and any fluid and electrolyte depletion is corrected. Vitamin K is administered, hemoglobin per cent estimated, and the patient's blood group ascertained. Administration of penicillin and streptomycin is commenced and morphia and atropine are given as premedication. Suitable operating conditions are obtained under general anesthesia using pentothal, nitrous oxide, oxygen, and flaxedil. The approach to, and exploration of, the bile ducts are the same as described earlier except to ensure that the distal end of the choledochostomy incision in the supraduodenal portion of the common bile duct is carried adjacent to the duodenum in anticipation of the anastomosis. Having steadied the duodenum with tissue forceps a transverse incision of about 2 centimeters is made in the anterolateral aspect of the viscus opposite to the opening in the common bile duct. Efflux of duodenal contents is slight, but brisk bleeding from the cut edge may tend to obscure the operation field and require temporary application of hemostats until insertion of the anastomotic suture. A side-to-side anastomosis between duodenum and common bile duct is now constructed using a single layer all coats suture, either continuous or knot-on-the-lumen interrupted. It is necessary to rotate the duodenum laterally somewhat in order to bring its anterior wall into apposition with the bile duct, and a relieving incision in the serous layer on its medial side may be helpful in this maneuver. The anastomosis is completed by tacking a piece of omentum over the suture line. In a few cases, early in the series, we additionally performed a cholecystostomy, when the gallbladder was left in situ, as a drainage safety valve. This step, however, has proved redundant as there is no tendency for immediate postoperative stenosis at the stoma and cholecystostomy drainage is minimal. Accordingly, we now omit it. Before closing the abdomen a split rubber tube and wick drain is placed down to the suture line via a separate stab incision and removed 48 hours later. Postoperative recovery is rapid and uneventful in uncomplicated cases. Gastric aspiration and parenteral fluids are continued for the first 2 days following which oral alimentation can be resumed successively with bland fluids and milk feeds, then soft diet up to about the seventh day, when normal meals can be

taken. Antibiotics are continued for 48 hours after the temperature becomes normal which occurs usually 3 to 4 days after operation. Jaundice decreases rapidly, to disappear within the first week.

During 1954, 19 cases of cholangiohepatitis were treated by choledochoduodenostomy. There were no operative deaths in this consecutive series, but 2 complications, wound sepsis and leakage from the anastomosis, were encountered in some patients. The latter occurred in 2 patients in whom bile started draining out along the drainage tube track 3 days and 5 days after operation respectively. Both biliary fistulas closed spontaneously, within a period of 1 month, the patients meanwhile being nursed prone on a Bradford frame to prevent efflux excoriating the skin which was further protected by applying Baltimore paste around the wound area. Wound sepsis proved troublesome in 4 patients and necessitated laying open the wound for drainage and healing by secondary intention. This latter complication was undoubtedly attributable to contamination of the wound by infected bile during the duct exploration. Clearance of calculi from the ducts is a protracted affair in many of these patients owing to the large amount of material to be removed, the necessity of ensuring as complete a clearance as possible, and the difficulties of dislodging the calculi, especially when intrahepatic. Under these circumstances there is ample opportunity for bile and debris to be spilled in the operation field, however vigilant the operator. The use of intra-abdominal packs effectively prevents spill into the remainder of the peritoneal cavity but it would seem to be equally necessary to protect the parietal incision with towels fixed to the cut edge of peritoneum as a precaution against contamination of the wound.

The remote postoperative progress of 16 of these patients has been followed up over a period of 12 months in the earliest and 3 months in the most recent case.

When last seen all patients were symptom-free and had remained so since their operations, with the exception of 1 patient who had a mild attack of fever and jaundice 1 month after discharge from the hospital but which subsided spontaneously within 5 days. All were in a good state of health and following their usual occupations. There was no trace of bile in the urine and serum bilirubin levels were in the range of normal.

FIG. 6. Barium meal roentgenogram taken with the patient in head down position. A small amount of barium can be seen entering the common bile duct which is outlined by contained gas.

DISCUSSION

In the treatment of cholangiohepatitis, choledochoduodenostomy seeks to ensure continuous free drainage of bile into the duodenum and so by eliminating stasis to allow the natural bodily defenses, aided by antibiotics, to overcome sepsis in the ducts. At the same time by short circuiting the hiatus of the ampulla of Vater, the creation of a new wide stoma permits recurrent deposits of biliary debris and any residual intrahepatic calculi, which may migrate, unhindered passage into the duodenum.

While opinion on the effectiveness of choledochoduodenostomy in preventing recurrence of cholangiohepatitis will have to be deferred until a much greater time interval has elapsed, certain conclusions may already be drawn. Quite outstanding is its advantage over the alternative surgical procedure of choledochostomy and T-tube drainage. It is certainly more acceptable from the patient's viewpoint to be discharged from the hospital with a completely healed abdominal wall than to tolerate a tube issuing from a sinus which requires periodic dressings. Again, it is suspected that leaving a tube in the common bile duct for a protracted length of time leads to foreign body irritation of the duct, promoting further damage and perpetuating infection. Furthermore, and interestingly enough, there appear to be no ill effects from by-passing the valve action of the ampulla with the attendant possibility of reflux of duodenal contents into the bile ducts.

In 14 patients on whom a barium meal series was carried out postoperatively, entry of the suspension into the biliary tract through the new stoma was seen to occur in 9 (Fig. 3). This, however, was only noted when the subject was positioned head down and lying either supine or on the right side. In no case was reflux seen with the patient upright, an indication that gravity and posture are the determining factors. Duodenal contents which enter the bile ducts readily drain back into the duodenum under the influence of gravity. A film taken with the patient sitting upright for half an hour after the roentgenogram illustrated in Figure 3 shows that during the interval the hepatic ducts have completely drained of contrast medium, a residuum of which is seen only in the common bile duct (Fig. 4).

Another consequence of establishing free continuity of the biliary and alimentary tracts by choledochoduodenostomy is the accumulation of intestinal gas in the intrahepatic ducts which feature is demonstrated in Figures 5 and 6.

One factor that would serve to explain the absence of untoward sequelae of reflux is that the duodenal contents, having passed through the antiseptic acid bath of the stomach, are normally sterile.

A feature arising in connection with cholangiohepatitis which is worthy of comment is the absence of any associated pancreatitis which we have not encountered in any of our patients. Obstruction at the duodenal ampulla with regurgitation of infected bile into the pancreatic ducts and activation of pancreatic trypsinogen as a cause of pancreatitis is a theory which has its adherents. The hypothesis does not, however, receive support from our own experience of cholangiohepatitis, attendant, it would seem, by just such circumstances as might be expected to lead this complication.

SUMMARY

1. The clinical and pathologic features of cholangiohepatitis as encountered in Hong Kong are described.

2. In our opinion liver fluke infestation, pyogenic infection, and obstruction of the bile ducts, either by spasm or inflammatory edema of the ampulla of Vater or impaction of biliary calculi, are the sequence of development of the syndrome.

3. Our experiences with the surgical management of the condition are described.

4. The postoperative progress of 16 patients treated during 1954 by choledochoduodenostomy is reviewed.

5. The rationale and merits of choledochoduodenostomy as compared with T-tube drainage of the bile ducts are discussed and the absence of associated pancreatitis with cholangiohepatitis is commented upon as well.

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THE EFFECT OF CHRONIC HYPERCHOLESTEROLEMIA ON CANINE AORTIC TRANSPLANTS

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WHEN A "DEAD" ARTERIAL HOMOGRAFT is transplanted to a "dying" arteriosclerotic aorta for the treatment of aneurysm or occlusive disease a unique biologic situation is created. The homologous artery, unlike other homospecific tissues, is accepted and eventually incorporated by the host (3). Thus, in its new environment there is the possibility that the vascular transplant will become susceptible to atherogenic factors in the host. This study was undertaken, therefore, to determine the effect of induced atherosclerosis on experimental vascular transplants.

METHODS

Atherosclerotic lesions have been produced in the dog by Steiner and associates (10, 11) and Bevans, Davidson, and Abell by the simultaneous oral administration of an antithyroid drug and cholesterol. When serum cholesterol levels of 1,000 milligrams per 100 cubic centimeters were maintained for prolonged periods, changes occurred in the blood vessels that closely resembled atherosclerosis in the human. In view of these observations, and because of the ease with which canine aortic transplantation is accomplished, dogs were chosen for these experiments.

Adult mongrel dogs weighing between 10 and 15 kilograms were anesthetized with nembutal sodium given intravenously and the abdominal aorta was exposed through a midline incision. After mobilization of the aorta below the renal arteries, a segment 1.0 to 1.5 centimeters long was resected and the resulting defect bridged with an aortic graft. The grafts were about 2.5 to 3.0 centimeters long and the distal suture lines were placed about 1 centimeter above the trifurcation.

In one group of animals homografts refrigerated in balanced salt solution were used, and in another group freeze-dried homografts. In a control group the abdominal aorta was mobilized, stripped of periaortic tissues, transected in two places, and the free segment was reimplanted. About 2 weeks following operation hypothyroidism was induced by parenteral administration of radioactive iodine in doses of 1 microgram per kilogram of body weight. The animals were then placed on a diet of horse meat (70 per cent), purina chow (25 per cent), and cholesterol (5 per cent), mixed in a tumbling type mixer. The degree of lipemia was followed by weekly determinations of serum cholesterol. The animals were sacrificed from 6 to 18 months after implantation of the homografts and after 5 to 15 months of cholesterol administration. At necropsy the heart and aorta with the graft were removed in continuity and fixed in formalin (10 per cent) for about 48 hours. The aorta was then opened longitudinally and the number and distribution of lesions in the aorta and graft were noted. Blocks of tissue were obtained for microscopic study from the graft and adjacent portions of the host aorta. Paraffin sections were prepared from each block and stained with hematoxylin and eosin, Masson's trichrome, and Weigert's elastic tissue stains. In addition, from selected portions

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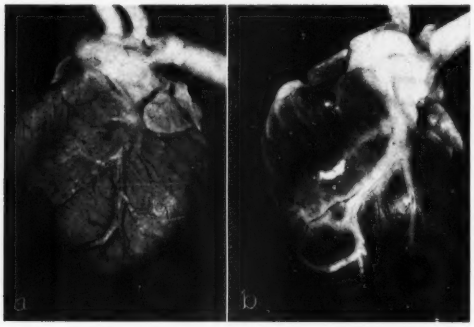


FIG. 1. Photographs of canine hearts illustrating: a, fixed and, b, fresh, demonstrating atherosclerotic plaques in the anterior descending branch of the left coronary artery.

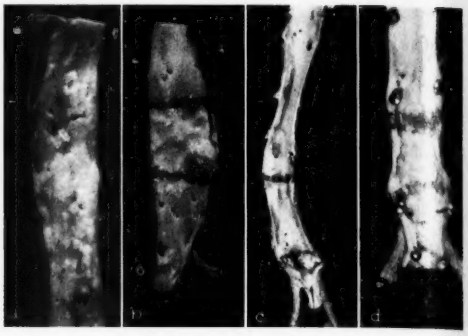


FIG. 2. a, Intimal surface of abdominal aorta, b, of a refrigerated homograft, c, of a freeze-dried homograft, d, of an autogenous graft. Raised yellow atherosclerotic plaques coalesce in places to cover inner surface.

frozen sections were prepared and stained with Sudan III.

RESULTS

The vascular changes produced by these experimental procedures were similar to those reported by Steiner and Kendall (10). The lesions were raised yellow-orange plaques 0.1 to 1 centimeter in diameter. In the coronary arteries the plaques produced beading, particularly along the anterior descending branch of the left coronary artery (Fig. 1). No lesions were noted in the ascending portion of the aorta. In the descending portion plaques were only around the orifices of the intercostal arteries. The lesions were more numerous in the abdominal aorta increasing in number and size toward the trifurcation and coalescing in places to involve the entire circumference. The lesions in the grafts were indistinguishable from those in the host vessels (Fig. 2).

The microscopic changes consisted of infiltrations with foam cells in the intima, media, and about vasa vasorum of the adventitia (Figs. 3, 4, and 5). In focal areas the media was broadened so that a bulge into the lumen was produced (Figs. 6 and 7). In these areas the elastic fibers were fragmented and replaced by an amorphous fibrillar substance and cholesterol clefts. These areas were infiltrated with large mononuclear cells having a foamy cytoplasm. In other areas atheromatous change in the intima was adjacent to cells with elongated nuclei in a whorl-like pattern (Fig. 8). Intimal plaques were usually associated with reduplication of the inner elastic lamina.

In animals with serum cholesterol levels of about 1,000 milligrams per 100 cubic centimeters atheromatous changes were invariably present in the coronary arteries, at the orifices of the intercostal arteries and in the abdominal aorta and graft. With lesser degrees of hypercholesterolemia there was diminution in the extent of these lesions, and in 1 animal with an average cholesterol level of 429 milligrams per 100 cubic centimeters no atherosclerotic change developed. Thus, there was a correlation between the level of hypercholesterolemia and extent of the atherosclerotic lesions. The duration of hypercholesterolemia beyond 5 months, however, did not appear to be a significant factor.

Of 26 animals surviving the experiment with patent abdominal aortic grafts, 13 had refrigerated homografts implanted into the aorta, 5 freeze-dried homografts, and 8 were controls with autogenous grafts.

Among the 13 animals with refrigerated grafts 5 had serum cholesterol levels in excess of 1,000 milligrams per 100 cubic centimeters (Chart 1). In these the grafts were implanted between 12 and 15 months and

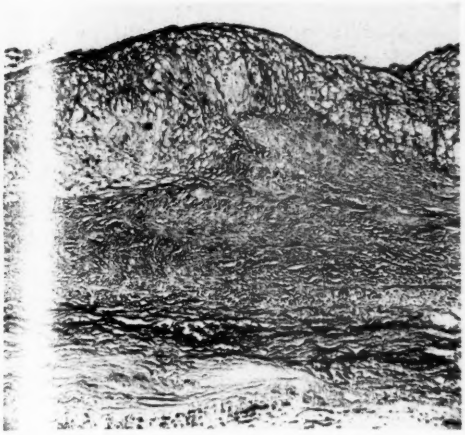


FIG. 3.

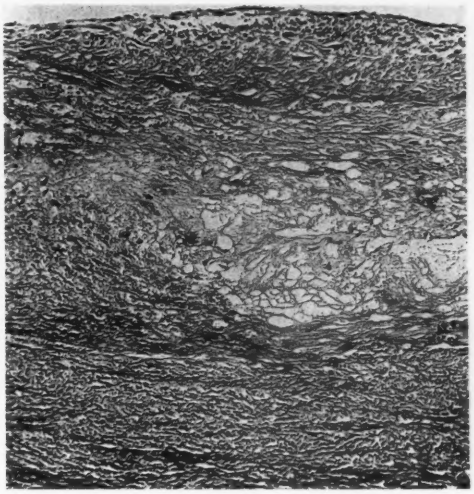


FIG. 4.

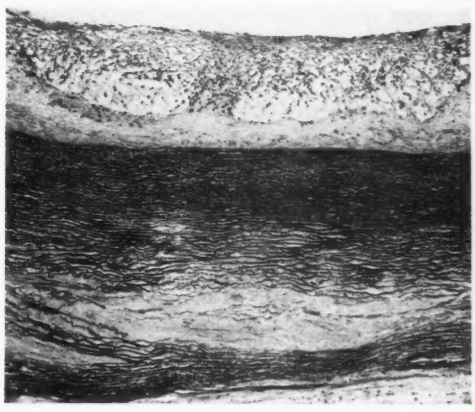


FIG. 5.

FIG. 3. Microscopic appearance of a refrigerated canine aortic homograft 12 months after transplantation and after 5 months of cholesterol administration. Beneath the intima and in the inner media are collections of large foam cells with eccentrically placed, compact nuclei. There is hyalin change in the adjacent media. Trichrome preparation, × 25.

FIG. 4. Microscopic appearance of an autogenous aortic graft 12 months after reimplantation and after 5 months of cholesterol administration. There are vacuolated areas in the media surrounded by foam cells. There is an increase of fibrous connective tissue in the overlying intima. Trichrome preparation, × 25.

FIG. 5. Microscopic appearance of a freeze-dried aortic homograft 16 months after implantation and after 13 months of cholesterol administration. The intima is broadened with acellular areas surrounded by collections of foam cells. Beneath is a zone of hyalinizing fibrous connective tissue. In the outer portion of the media there is fragmentation and disappearance of the elastic fibers. Weigert preparation, × 25.

cholesterol had been administered for 5 months when the experiment was terminated. In each instance the host aorta and trifurcation and the homograft were equally involved in the atheromatous process: in 4 to a maximum degree, and in 1 to a lesser extent. In the remaining 8 animals the average serum cholesterol levels ranged between 429 and 776 milligrams per 100 cubic centimeters (Chart 2). When the experiment was terminated 17 months after operation and after 8 months of cholesterol administration 4 of the animals had no atherosclerosis in the host vessels, and 4 had only minimal changes. In the homografts, on the other hand, there were atheromatous changes in 7. In 2 of these the lesions were extensive and in 2 moderately extensive. Thus, in animals with refrigerated homografts having serum cholesterol levels above 1,000 milligrams per 100 cubic centimeters the host aorta and graft were equally involved; whereas, in those with serum cholesterol levels less than 1,000 milligrams per 100 cubic centimeters the

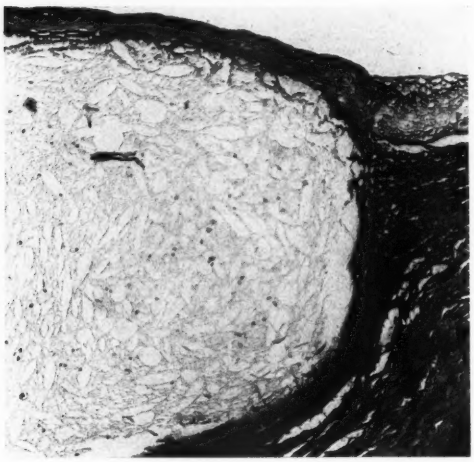


FIG. 6. Microscopic appearance of a refrigerated aortic homograft 12 months after transplantation and after 5 months of cholesterol administration. The inner elastic lamina is reduplicated and the adjacent elastic fibers of the media are widely spread apart by an atheromatous plaque containing cholesterol slits and foam cells. Weigert preparation, × 80.

homografts appeared to be selectively affected by the atherosclerotic change.

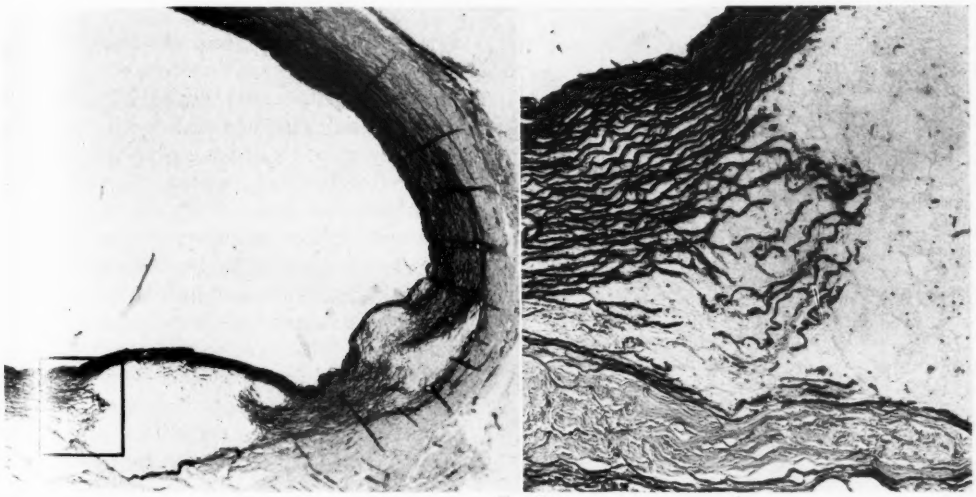
Among 5 animals with freeze-dried homografts the average serum cholesterol level ranged between 476 and 935 milligrams per 100 cubic centimeters (Chart 3). At the termination of the experiment 15 to 18 months after operation and after 13 to 15 months of cholesterol administration minimal atherosclerotic changes were noted in the host aortas in 3 animals and the aortas were normal in 2. In contrast, there were atherosclerotic changes in all of the homografts and in 3 the changes were extensive. Thus, the freeze-dried homograft also appeared to be selectively affected by the atherosclerotic change in animals with average serum cholesterol levels less than 1,000 milligrams per 100 cubic centimeters.

Among the 8 control animals with autogenous grafts there were 6 with average serum cholesterol levels between 1,008 to 1,746 milligrams per 100 cubic centimeters and 2 with levels of 609 and 563 milligrams per 100 cubic centimeters respectively (Chart 4). At the termination of the experiment 7 to 12 months after reimplantation of the grafts and after 5 to 8 months of cholesterol administration the 6 animals with high serum cholesterol levels disclosed atheromatous change involving the host aorta and autogenous graft equally. In 2 the changes were extensive, and in 4 moderately extensive. In 2 animals with low serum cholesterol levels no atheromatous changes were noted in either the aorta or the graft. Thus the autogenous graft and the host vessel appear to respond alike to varying degrees of hypercholesterolemia.

DISCUSSION

It is generally agreed that atherosclerosis is a result of local vascular and systemic metabolic alterations. Intramural hemorrhage as a local factor contributing to the development and progression of atherosclerotic lesions has been emphasized by Winternitz (14, 15) and Paterson. Small hemorrhages into proliferative areas in the wall of the vessel are followed by a gradual accumulation of cholesterol and blood pigment, and the formation of fibrous connective tissue. The effects of intramural hemorrhage are due in part to organization of the thrombus, to deposition of cholesterol and other solid elements, and to tissue reaction to these materials. Vascular granulation tissue is thus formed and a vicious cycle is initiated in that hemorrhage stimulates repair and repair predisposes to further hemorrhage.

The changes occurring in the elastic tissue elements within the media are thought to be important in the development of atherosclerosis. There is a reduced capacity for distention of the vessel wall during cardiac systole and recoil during diastole with resulting elongation and dilatation of the vessel. According to Hass this change results from discontinuities in aged elastic tissue rather than a reduction in the property of elasticity. On the other hand, Lansing has noted a change in the chemical composition of elastic tissue in the media of the aorta and pulmonary artery of patients with athero-



a b

FIG. 7.

FIG. 7. Microscopic appearance of a refrigerated aortic homograft 15 months after transplantation and after 6 months of cholesterol administration. There are two atheromatous plaques where the continuity of the elastic fibers is interrupted. In the inset the atheromatous change is composed of cholesterol slits and foam cells. The elastic fibers are spread apart or are absent in places. Weigert preparation, a, × 13; and b, × 85.

FIG. 8. Microscopic appearance of a freeze-dried aortic homograft 18 months after transplantation and after 15 months of cholesterol administration. There is an atherosclerotic plaque in the intima composed of hyalinizing fibrous connective tissue with infiltration of lymphocytes, plasma cells, and large mononuclear cells near the surface and of foam cells beneath. The plaque extends into the adjacent media disrupting the elastic fibers. Weigert preparation, × 25.

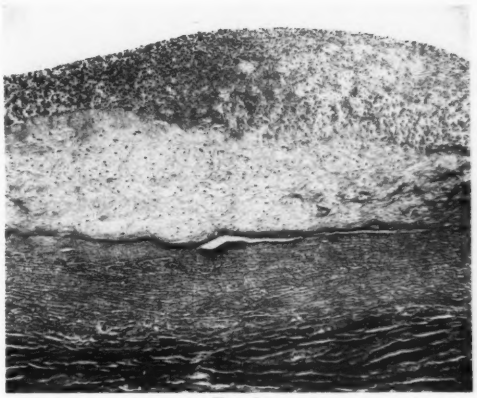


FIG. 8.

sclerosis. Whatever the cause, when these changes are generalized the systolic blood pressure becomes elevated to compensate for the loss of arterial elasticity and the pulse pressure is increased to maintain physiologic requirements. Thus arteries with reduced functional elastic elements are subjected to greater pressures in order to attain adequate arterial distention and recoil. This places greater stress upon the vessel wall, and new sites of injury and repair are likely to develop.

Still another mechanism by which changes in the elastic media may contribute to the development of atherosclerotic changes has been reported recently by Taylor (13). He demonstrated that lesions of atherosclerosis are associated with cracking or fraying of elastic fibers and by histochemical methods identified a substance at these sites as a mucopolysaccharide. It is suggested that degenerative changes in the elastic tissue elements of the media result in deposition of mucopolysaccharides which in turn appear to stimulate the development of atherosclerotic lesions.

Finally, Duff has pointed out an anatomic feature of arteries that may explain the predilection of arterial bifurcations for the de-

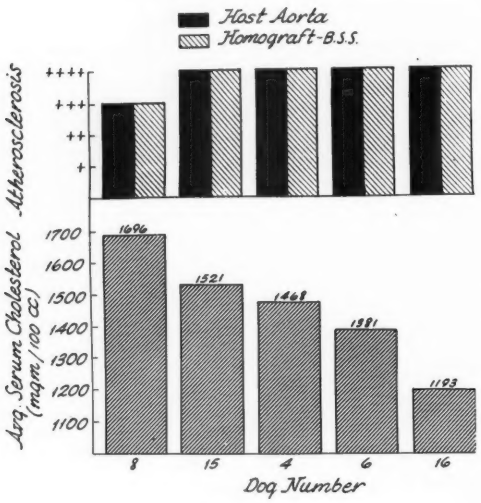


CHART 1.

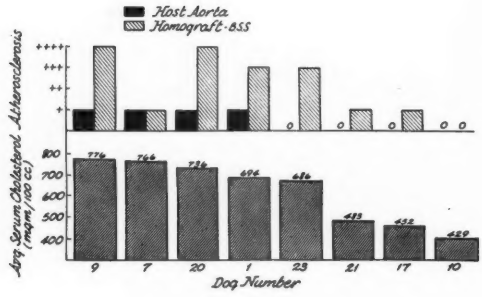


CHART 2.

velopment of atherosclerosis. He states that at points of bifurcation, just before division into two branches occurs, the lumen of the parent artery tends to widen. In a blood vessel of variable caliber that is filled with blood under constant pressure, the tensional stretching force in the wall of the vessel is greater in the wider part than in the narrower parts. Therefore, if the wider part of the artery is not equipped with a thicker, stronger wall than the rest, it will stretch and consequently be injured more readily. Since widening of the lumen just proximal to bifurcations is not associated with a protective thickening of the vessel wall, these areas are subject to a stretching force greater than that which occurs elsewhere and become sites of predilection for the development of atherosclerotic change.

It is now recognized that the transplanted homologous arterial graft rapidly loses its structural identity and is replaced by fibrous connective tissue elements of the host, only the elastic tissue surviving (3, 4). Of the structural changes which the homograft undergoes, two are of particular significance. First, the gradual conversion of the graft to a connective tissue tube is brought about by the formation of intramural granulation tissue abundant in new capillaries. These rupture readily, and the resulting extravasations of erythrocytes stimulate further connective tissue growth. Under some circumstances, however, bleeding may be excessive and may result in minute hematomas within the walls of the transplant. The calcific deposits in the wall of some homografts one or more years of age probably represent areas of such prior hematomas. Secondly, the elastic fibrils lose some of their wavy contour and become disrupted and fragmented. The waviness of elastic fibers in the media is believed to be responsible for their spring-like action, permitting distention and recoil. In addition to these two factors occurring as a natural part of aging of homologous arterial tissue, a third factor—widening of the arterial lumen in the region of the graft—may result from a disproportion between graft and host vessel existing at the time of transplantation. Although in most instances of disproportion the graft is smaller than the adjacent vessel, in some instances the diameter of the homograft exceeds that of the recipient vessel. Thus, homologous arterial transplants exhibit naturally some of the local vascular changes considered essential to the development of atherosclerosis.

Although the number and scope of these preliminary experiments are too limited to justify conclusions, it appears that the canine homograft is susceptible to the metabolic atherogenic factor of hyperlipemia. Moreover, when serum cholesterol levels are only moderately elevated above normal the ho-

mografts seem to be selectively involved in the atheromatous process. Thus of 13 animals with serum cholesterol levels less than 1,000 milligrams per 100 cubic centimeters only 7 had atheromatous lesions in the aorta, all slight; whereas all but 1 of the homografts were affected, 7 severely.

Similar results were noted by Gentsch and associates in acute experiments. They implanted freeze-dried aortic homografts into the thoracic aorta of dogs and the animals were then subjected to hypervolemic lipid stress. When examined 28 to 86 days later, all of the grafts were extensively infiltrated with lipids, foam cells, and inflammatory cells and were more extensively involved than the adjacent host aorta.

Experiments by Taylor, Baldwin, and Hass (12) and by Baldwin and associates (1) also appear to support our observations. In comparing the effect of arterial injury in normal vegetarian and hypercholesterolemic rabbits, these investigators observed that under normal conditions arterial injury was followed by formation of a new arterial wall within a proliferating intima, whereas under conditions of hypercholesterolemia a high incidence of localization of lipids or atheromatous deposits was noted at sites of injury.

No attempt was made to correlate the sites of development of atheromatous lesions with specific changes occurring in the walls of the homografts as part of the aging process. The failure of two autogenous grafts to develop atherosclerosis when the serum cholesterol was less than 1,000 milligrams per 100 cubic centimeters suggests, however, that the structural changes taking place within the graft are significant. The autogenous transplant undergoes little structural alteration (4), whereas the transplanted homograft is almost completely transformed by tissues of the host.

In these experiments the grafts were implanted into the abdominal aorta, a site of maximal involvement in the hypercholesterolemic dog. However, if the homograft is selectively affected by hyperlipemia, grafts implanted into the thoracic aorta should be affected by the atheromatous process about equally with grafts in the abdominal aorta. This and other aspects of the problem are now being investigated.

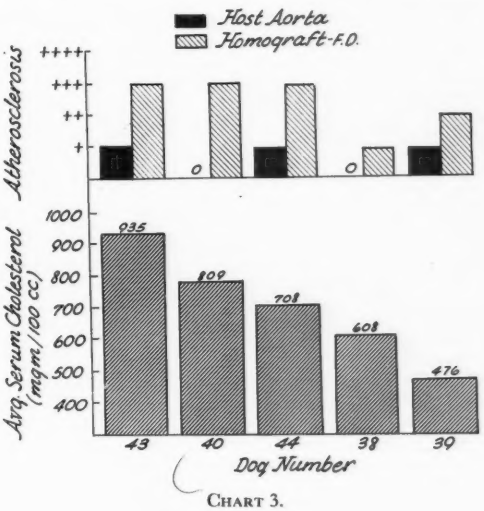


CHART 3.

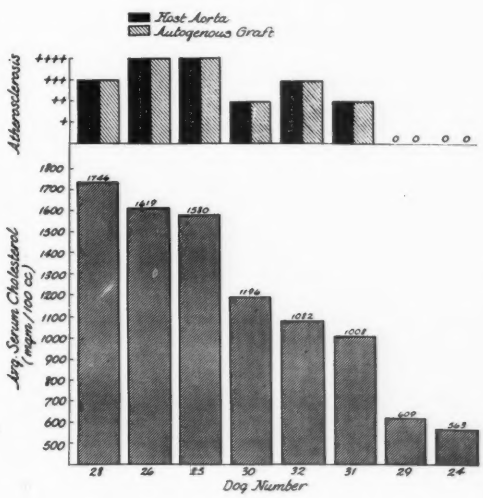


CHART 4.

SUMMARY

The effect of chronic hypercholesterolemia on canine aortic transplants was studied. Following implantation of aortic homografts into the abdominal aorta, the animals were

rendered hypothyroid by administration of radioactive iodine. Cholesterol was then added to the diet to produce hypercholesterolemia. A control group with autogenous aortic grafts was similarly treated. When sacrificed 6 to 18 months after transplantation of the grafts, the host aorta and homografts were equally involved by atherosclerotic lesions in animals with average serum cholesterol levels in excess of 1,000 milligrams per 100 cubic centimeters. In animals with serum cholesterol levels less than 1,000 milligrams per 100 cubic centimeters there was only minimal or no involvement of the host aorta, whereas in every instance the homograft was moderately to extensively involved. In the animals with autogenous aortic grafts and with serum cholesterol levels above 1,000 milligrams per 100 cubic centimeters the aorta and graft were equally involved; whereas in those with lower serum cholesterol levels the grafts were unaffected.

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FREEZE-DRIED ARTERIES USED AS TENDON SHEATHS

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IN THE FIELD OF TENDON SURGERY, a major problem is the prevention of adhesions and maintenance of free gliding motion. Evidence is available to suggest that the adhesions are initiated by the paratenon and surrounding tissues and not by the tendon itself. The procedures that have been designed to prevent these adhesions operate in one of two ways. In one, a new smooth-walled tunnel is allowed to form around a tubular mold to provide a gliding surface for the tendon after the mold is removed. In the other procedure, the tendon is sheathed during healing with a substance that is less likely to adhere to the tendon than surrounding connective tissue.

The main proponents of the method of cannalization have been Mayer and Ransohoff. They implanted molded celloidin tubes in the tendon area and allowed them to remain from 4 to 6 weeks. After the tube was removed, a tunnel with a smooth, flat, fibrous tissue surface remained. An autologous tendon graft could be placed within this new tunnel. They chose celloidin because of its plasticity and because it incites little fibrous tissue reaction. However, the preparation of the mold was time consuming. Thatcher used this method but employed stainless steel rods, implanted for 3 weeks. Tygon tubes were used by Hanisch and Kleiger to produce tendon channels in the same manner. Although all men reported encouraging results both experimentally and clinically, a disadvantage intrinsic in the cannalization method is the necessity of two operations.

Numerous substances have been interposed between the tendon and the surrounding tissues. They can be roughly classified into two large groups, inorganic and organic.

The inorganic materials include sterile vaseline and rolled silver tubes, cellophane, rubber latex, tantalum foil, nylon, tygon, polyethylene, and thin stainless steel foil. All materials were tried in animals and some were used clinically with moderate success. The adhesions and fibrous scarring around the tendon were not consistently prevented by any of these substances, except polyethylene. However, with this material, the time required for tendon healing was doubled.

The group of organic materials is large and includes substances of autologous, homologous, and heterologous origin. Autologous fat, fascia, and tunica vaginalis were not successful unless there was early mobilization of the tendons. Homologous cartilage and homologous vein preserved in formalin were tried unsuccessfully. The tissues of heterologous origin were beef amniotic membrane, allantoic membrane, and the mucosa of beef cecum. None proved satisfactory.

Cargile membrane, chromicized pig's bladder, was used by Henze and Mayer in 1914. Although they had some success with it in animals they concluded that "autoplastic transplants, preserved animal tissues, or foreign bodies introduced as an artificial sheath, tended to exaggerate rather than to inhibit the adhesions." Sheet catgut, used by Davis and Aries, also was a failure. A material called amnioplastin, prepared from human amnion, was used by Pinkerton in 1942. He reported 4 clinical cases with good results. Weckesser and associates tested human fibrin film, bovine fibrin film, gelfoam, and oxycel cotton. Again, none was satisfactory.

Homologous vascular tissue to prevent adhesions about a suture line was first used by

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The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

FIG. 1. Arterial homograft sheath after 6 months. It has been split longitudinally and the tendon retracted. Note the smooth, shiny, gliding surface and the absence of adhesions.

Huber in 1920. He wrapped a formalinized and alcohol-treated artery around a repaired nerve. Results were inconclusive and he decided that healing was the same with or without the vascular sheath. In 1943, Weiss observed that fibrous tissue was prevented from penetrating a freeze-dried homologous arterial sleeve which was placed around a severed nerve.

The preliminary work with freeze-dried arteries to prevent adhesions to tendons was done by Richard S. Oakey in this Institute. In his experience, autologous and fresh homologous vein grafts as well as fresh autologous arterial grafts failed to prevent adhesions. In one leg of each of 10 dogs he placed an open freeze-dried arterial segment between a damaged tendon and the skin. The tendon of the other leg was damaged in the same manner, but no graft was inserted and served as a control. Adhesions were prevented in all cases by the interposed freeze-dried homologous arterial segment, while adhesions formed in all the control cases.

With the exception of freeze-dried tissue, the organic materials did not consistently prevent adhesions to the tendon or provide an adequate plane of cleavage for tendon motion. In view of the disadvantages and inconsistent results of previously reported methods and because of the early success in preventing adhesion with the freeze-dried arteries, a more extensive study of the use of this material in the field of tendon surgery was undertaken.

METHODS

Animals. Thirty-six healthy mongrel dogs weighing between 15 and 20 kilograms were used. All received the same standard diet and were housed in heated rooms. They received 300,000 units of procaine penicillin, and 0.5 gram of streptomycin on the day of each operation.

Graft material. The arteries were taken at sterile autopsy, or were sterilized with the vapor ethylene oxide technique after routine autopsy. After they were frozen in an alcohol and dry ice mixture, the free water was removed by sublimation in a vacuum of approximately 50 microns. The arteries were then sealed in a vacuum in their original containers and stored at room temperature. Before use, the normal physical properties of the blood vessel were restored by reconstitution in sterile normal saline. The graft was then implanted using aseptic technique.

PROCEDURE AND RESULTS

The first phase of the experiment was designed to determine, in simplest fashion, whether a freeze-dried arterial homograft

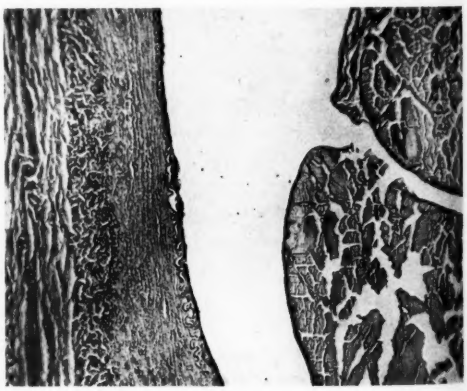

FIG. 2. The tendon is on the right; the arterial sheath is on the left. Note the lack of adhesions and the smooth flat cell layer on the surface of the tendon and the sheath. The shattering of the tendon is an artifact caused by the histologic preparation. Dog sacrificed at 7 months. Hematoxylin and eosin stain, ×11.

used as a sheath for a traumatized tendon actually would prevent or minimize the formation of adhesions between the tendon and surrounding structures. The test was made on 14 dogs in each of which both forelegs were used. Trauma to the tendon consisted of removal of all tissue that permits smooth, gliding motion. The paratenon was dissected away, the mesotenon was divided, and the peritenon was scraped from the tendon. A 3 centimeter segment of freeze-dried dog aorta or carotid artery was placed around the traumatized portions of the extensor digitorum communis tendon in 19 legs. In 5 control legs the trauma to the tendon was the same, but no graft was inserted. No attempt was made to immobilize the legs in any of these animals. The dogs were observed from 3 weeks to 14 months.

Normal motion was maintained with 16 of the homograft arterial sleeves and was slightly limited in 4 control legs. Three of the sheath sites and 1 control site became infected with destruction of the graft and tendon. Gross and microscopic results were excellent with 11 of the grafted sleeves and good with 5 (Fig. 1). Those considered excellent showed no adhesions and had a shiny, flat connective tissue cell layer over the internal surface of the implanted artery (Figs. 2 and 3). Those considered good were similar, except that adhesions were present at the extremes of the graft where the destruction of the paratenon and the peritenon had extended beyond the graft site. All of the freeze-dried arterial implants were replaced with host fibroblasts. The noninfected control legs showed adhesions to the tendon, but only slight limitation of motion. The fibrous bands had stretched because of the self-administered physiotherapy.

Since the freeze-dried arterial homograft appeared to function adequately as a tendon sheath, the second phase of the project was designed to simulate the conditions under which a homologous pseudosheath might be used clinically. This required a three stage procedure. First, it was necessary to produce a densely adherent and scarred tendon. This

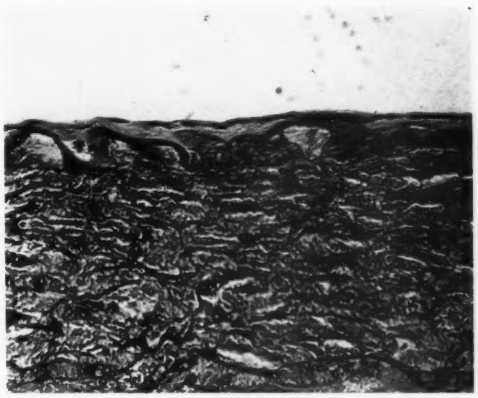

FIG. 3. Higher magnification of the flat connective tissue cell layer on the surface of the arterial sheath. Note the elastic lamellae. Orcein stain, ×73.

was accomplished by splinting the leg after trauma and partial division of the tendon. One foreleg in each of 14 dogs was used. The extensor carpi radialis was the tendon selected, since it is one of the largest and longest of the tendons. It is surrounded by paratenon as well as a true sheath at the distal portion, where it lies beneath the abductor pollicis longus. This latter muscle was severed in its tendinous portion to expose the extensor carpi radialis.

Stage 1 consisted of the partial division and suture of the tendons with destruction of all the gliding tissues and traumatization of the underlying periosteum. The legs were immobilized at both the humeroradial and radiocarpal joints with Kirschner wires. Approximately 10 to 14 days after stage 1 the tendon was found to be firmly bound down to the surrounding tissues and bone. This observation at the second stage operation served as the control. At this time, the tendon was freed and an arterial graft sutured around the raw portion as a sleeve. The immobilization was continued until stage 3, which consisted of the removal of the Kirschner wires about 2 weeks later. The dogs were sacrificed at 15, 30, 45, 60, and 90 days after graft implantation.

Three of these 14 legs became grossly infected and the grafts and tendons were de-

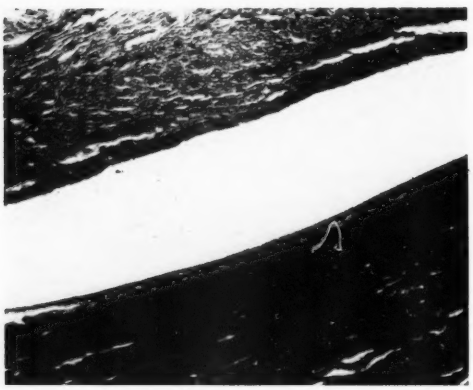

FIG. 4. Photomicrograph of a section through the tendon (top) and sheath (bottom) at the healing fibrous junction of the cut tendon. The leg was splinted for 2 weeks after implantation of the graft around the freed, bound tendon. Active motion was allowed for 2 days before sacrifice. Verhoeff's stain, ×36.

stroyed. All the dogs developed some adhesions between the tendon and the surrounding tissue through the lateral suture line of the sleeve. In 7 dogs, bands of fibrous tissue grew in through holes in the grafts. In 3 dogs the holes resulted from arterial branches that were cut flush and not repaired; in 2 dogs the holes were caused by localized areas of infectious destruction of the graft; and in another 2 dogs both types of defect were present.

At examination before sacrifice normal motion was possible in all the legs in which the arterial graft and tendon were not destroyed by gross infection. The few, thin adhesions that formed through the suture line and the holes in the graft did not restrict normal motion. Where intact, the arterial sleeve prevented any adhesions from forming between the tendon and the surrounding soft tissue or the underlying bone (Figs. 4 and 5).

If these results are to have clinical application, there arises the problem of supply of human arterial homografts. Since heterologous arterial sleeves would be more readily available for this use, an attempt was made to ascertain whether heterografts would also prevent adhesions to the tendons. Therefore, another series of 10 dogs was studied. The tendon of one foreleg of each dog was traumatized and a section of calf carotid artery was used as the arterial tendon sheath. Immediate use of the legs was permitted. The dogs were observed from 3 weeks to 10 months. There was whole or partial destruction of the graft with firm adhesions between the tendon and arterial sleeves in every instance. This failure is considered to be the result of marked host tissue reaction to the heterologous implant (Fig. 6).

DISCUSSION

We believe the material used around a tendon to prevent adhesions should: (1) be replaceable by host fibroblasts, (2) have the property of preventing these fibroblasts from penetrating through to the tendon before the raw surfaces are healed, and (3) not contribute to or stimulate excessive fibrosis in the operated area. The observations of Pate and Weiss showed that freeze-dried homologous arteries demonstrated the properties mentioned.

Following trauma and repair, a tendon normally becomes swollen and a poor result is obtained if it is constricted by a tough fibrous tunnel similar to the flexor tunnels in the finger. Since the artery is an elastic tube, it would be expected to accommodate this swelling, and not cause pressure necrosis. In 3 dogs that were explored 15 days after the sleeve implantation, the partially severed

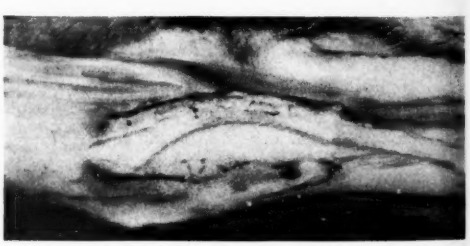

FIG. 5. No adhesions between a healed tendon and arterial homograft sleeve after 7 months. This tendon was partially severed and the leg splinted for 2 weeks before tendolysis. After placement of the sleeve, immobilization was maintained for another 2 weeks.

tendon was observed to be swollen and edematous but not constricted. Microscopically, the tendon appeared to be healing quite normally.

Since the freeze-dried artery is replaced by host fibroblasts, it is not a permanent barrier between the surrounding tissues and the new gliding surface that is formed. As this homograft artery becomes part of the host, it allows continued nourishment of the living cell lined gliding surface that has been established. In contrast, however, the inorganic materials remain as a permanent foreign body. Organic membranes other than freeze-dried homologous tissue incite a prolific fibroblastic host reaction, with resultant adhesions.

Two important points of technique must be emphasized. The suture line of the arterial sleeve should be as nearly perfect as possible to prevent the ingrowth of fibroblasts. The branches of the blood vessel graft must be tied for the same reason. Although the physical properties of the artery are regained after reconstitution, the tissue is dead. The implant has no active resistance to infection and is destroyed when gross infection occurs. Therefore, a good clinical result cannot be expected if the graft is implanted in a heavily contaminated area.

Adequate blood supply is necessary for good tendon healing. Skoog and Persson have shown that healing of a divided tendon is delayed if the site of division is completely and permanently isolated from the surrounding tissues by a tube of stainless steel foil. Gonzales reported also that healing was considerably delayed if the tendon was sheathed with polyethylene, but strong union did occur with long periods of immobilization. No new mesotenon is formed with the use of the freeze-dried arterial sheath; however, there is no interference with the vascular supply carried within and on the surface of the tendon. No attempt was made in this series of experiments to evaluate the healing of a completely severed and repaired tendon within the grafted sleeve. This problem is now under study.

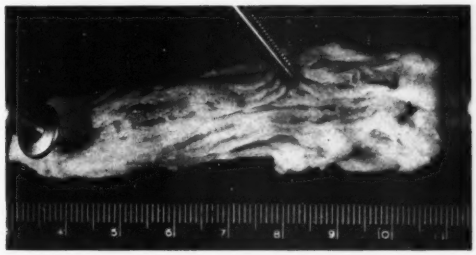

FIG. 6. Gross, firm adhesions between the heterograft arterial sheath and tendon after 6 months. The sheath has been incised longitudinally but cannot be removed from the tendon.

Although the extensor tendon of the dog is not the perfect counterpart of the flexor tendons in man, we believe this tendon area is adequate to test the ability of the freeze-dried material to prevent adhesions.

In spite of these possible objections, it would appear at this time that a freeze-dried arterial sleeve will prevent adhesions after tendolysis of a healed and adherent tendon in a reconstructive surgical procedure.

SUMMARY

Freeze-dried homologous arterial implants, when placed around traumatized tendons as a sleeve, prevented adhesions between the tendon and the surrounding tissue. The implant was replaced by fibroblasts, and a new gliding surface was provided for the tendon.

Freeze-dried heterologous arterial implants, used in the same manner, were not successful because of excessive fibrosis and scarring.

In the presence of infection, the freeze-dried material was wholly or partially destroyed.

This method of preventing adhesions between a tendon and the surrounding tissues may be a useful adjunct to the clinical techniques of tendon surgery.

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THE SURGICAL TREATMENT OF BRANCHIAL CYSTS AND FISTULAS

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FROM TIME TO TIME articles have appeared in the literature dealing with anomalies arising from the branchial apparatus. Few of these record sufficient experience to justify conclusions regarding the clinical behavior of branchial anomalies. This presentation is devoted to a review of 90 examples of branchial cleft cyst and fistula. Particular attention is given to agreement or disagreement with the characteristics of the anomalies as stressed by previous authors.

EMBRYOLOGY

In 1825 Rathke described branchial clefts in human embryos, and since that time there has existed only a rather vague understanding of the branchial apparatus. This limited understanding is due partly to the paucity of case material available for study.

During the second and third week of embryonic life there appear, in the region of the future neck, paired sets of branchial arches (aortic arches), of branchial clefts, and of pharyngeal pouches, a system resembling closely the permanent gill arches and gill clefts of fish. The branchial arches, numbering 5 and sometimes 6, are separated by the branchial clefts. Each cleft is in contact with an outpouching of the primitive pharynx, the pharyngeal pouch, and the two structures are separated from one another by a thin mesodermal membrane. This complex arrangement is unique, since derivatives of the three germ layers, ectoderm (cleft), endoderm (pouch), and mesoderm (arch), are concentrated in the small cervical area. It is not surprising then that branchial cysts and fistulas arising in close proximity to these germ layers may be lined either by squamous (ectodermal) or at other times by columnar (endodermal) epithelium. Nor is it difficult to appreciate that countless possibilities for developmental errors exist, explaining in part the occasional unusual location of these lesions and explaining, as well, the occasional finding of thymic tissue within these anomalies. For the same reasons, these anomalies have been referred to by a multiplicity of terms: embryonal inclusion cyst, pharyngothymic cyst, embryonic thymic cyst, epidermoid cyst, sebaceous cervical cyst, lateral cervical cyst, cervical cholesteatoma, branchial dermoid, sequestration dermoid, branchiogenic or branchial cyst. We prefer the term branchial cleft cyst which indicates the origin of these anomalies from the branchial clefts.

Some investigators attribute the origin of branchial cleft fistulas to the mere rupture of the thin mesodermal membrane between cleft and pouch. By this mechanism a communication of the primitive pharynx with the exterior may be established. The transitory embryonic branchial apparatus undergoes numerous transformations which eventually terminate in the definitive structures of the human adult. The consensus is that the first pharyngeal pouch gives rise to the eustachian tube, middle ear, and mastoid cells; the second pouch gives rise to the supratonsillar fossa; the third pouch gives rise to the thymus gland and upper paired parathyroid glands; the fourth pouch gives rise to the lower paired parathyroid glands and contributes to the formation of the thyroid gland. The derivatives of the first

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branchial cleft are the external auditory canal and the membrane between cleft and pouch forms the tympanic membrane. Since most branchial cleft fistulas communicate with the supratonsillar area of the pharynx, it is believed that they arise from the second branchial cleft.

The paired branchial arch contains a branch of the ventral aorta (aortic arches), cartilage, and a nerve. Eventually, most of these arches disappear. It is believed that the first and second arches disappear, but the anterior border of the first arch is represented by the facial artery and that of the second arch by the lingual artery. The first or mandibular arch contains a superior (maxillary) and inferior (mandibular) component. The former gives rise to the cheek, the maxilla, and the lateral parts of the upper lip; the latter gives rise to the mandible, cheek, and lower lip. Contributing to the formation of the external ear are portions of the first and second branchial arches. Hence, it is believed that preauricular sinuses are in no way associated with branchial clefts, but represent developmental errors arising from faulty fusion of the respective components of these two branchial arches. The nerve contained within the first branchial arch is the mandibular division of the fifth cranial nerve which supplies those muscles arising from this arch. The second branchial arch (hyoid arch) gives rise to the lesser wing of the hyoid bone, the stapes, and the styloid ligament. Within this arch is found the facial nerve. The latter supplies the muscles of facial expression which are distributed widely throughout the head and neck. The third arch gives rise to the lesser wing of the hyoid and its nerve is the glossopharyngeal. The nerves contained within the fourth and fifth branchial arches are the superior laryngeal and the inferior laryngeal branches of the vagus. The third branchial arch persists as the first portion of the internal carotid artery, while the fourth branchial arch gives rise to the first part of the right subclavian artery, and on the left, it gives rise to part of the arch of the aorta.

PATHOGENESIS

There can be no doubt that branchial cleft cysts and fistulas are developmental anomalies arising in association with these growth processes. This explanation accounts for the inconsistency of location and pathologic picture which is seen occasionally. Indeed, this very inconsistency regarding location and pathologic pattern is proof, if proof be needed, that these conditions are developmental rather than genetic in origin, a point substantiated by the statistics of this review inasmuch as we were unable to find 1 instance in which a familial history could be elicited. Most branchial anomalies lead from a point somewhere along the anterior border of the sternocleidomastoid muscle to the upper pharynx, thereby further fortifying the concept that they originate in association with the second branchial cleft. Wenglowski carried out extensive and brilliant studies on the embryology of these anomalies but concluded inaccurately that these developmental errors originated in the third pharyngeal pouch and represented a persistent thymic duct. Wenglowski was influenced to a great degree by the fact that thymic tissue is sometimes included within branchial cysts.

Recently, as would be expected, branchial cysts and sinuses associated with the first branchial cleft have been described although there are no instances on record of branchial anomalies originating from the third and fourth branchial clefts, a situation which would be theoretically possible.

DIAGNOSIS

A sinus diagnosed within the first year of life is almost always branchial in origin. No further investigation is needed for a diagnosis but lipoidal studies of the tract are desirable for in this way the extent of the sinus can be determined and the diagnosis of fistula may be established. Dye in the neighborhood of the external auditory canal would make the clinician suspicious of the origin in the first branchial cleft. A child bearing a cyst in his neck presents a difficult problem in diagnosis. Often the differentiation between bran-

FIG. 1.

FIG. 1. A branchial cleft cyst in a 17 year old colored female.

FIG. 2.

FIG. 2. A branchial cleft sinus (arrow) directly over the lower third of the sternomastoid muscle.

branchial cleft cyst and cystic hygroma is impossible until the neck is explored. A common source of diagnostic error is an infected lymph node, and less frequently, one must seriously consider the lymphoid tumors. Tumors of the parotid gland are practically never seen at an early age, a point emphasized by the authors in recent publications (2, 6). Metastatic tumors are rare in infancy.

In the adult, greater diagnostic difficulty is encountered. Branchial cleft cyst in adults becomes a matter of differential diagnosis of tumors of the neck.

Metastatic carcinoma. The most frequent cause of a mass in the neck is metastatic carcinoma. This possibility must always be borne in mind and can be eliminated only when a thorough search for a primary carcinoma has failed to reveal evidence of carcinoma elsewhere. A careful search should be made for carcinoma within the nasopharynx, mouth, and larynx.

Tuberculous adenitis. This condition, although of infrequent occurrence, can present a difficult problem in diagnosis. It may occur at any age. A tuberculous lymph node may be firm and nontender, especially if it has undergone calcification. Secondly, with accompanying infection, it may be tender and fluctuant. Characteristically, a mass of tuberculous nodes is firm and fixed to surrounding tissues by fibrous reaction. Lastly, caseation may lead to sinus formation. Lymph nodes undergoing caseous necrosis with sinus formation may present an extremely difficult problem because their content may simulate the material found within branchial cleft cysts. In both conditions, the contents are sterile. Noteworthy of mention is that the material within the branchial cleft cyst is high in cholesterol crystals; consequently, if this fluid is left standing, a lipoid character is observed. This was pointed out as early as 1855 by Langenbeck and re-emphasized by Bailey in 1922. However, it should be borne in mind, that pseudocysts produced by liquefaction and by necrosis of solid masses may contain a substance high in cholesterol.

Primary tumors of the neck. Excepting thyroid carcinoma, these are, for the most part, lymphoid tumors, a term which has come to include tumors originating from any cell type found within a lymph node. Thus a complex series of tumors are encountered having as their origin any type of cell varying from the mature small lymphocyte to the more undifferentiated reticulum cell. Included are the more benign follicular lymphomas, the

malignant small cell lymphosarcomas, the more malignant large cell lymphosarcomas and the highly malignant reticulum cell sarcomas. These tumors practically never occur in the midline and are worthy of consideration in differential diagnosis only when found in the lateral regions of the neck. Lymphoid tumors more commonly involve groups of nodes and tend to be multicentric in origin. Finally, Hodgkin's disease must be included in this group of tumors. Aspiration yields information as to whether a given tumor is cystic or solid but pathologic diagnosis of cellular detail based on aspiration biopsy often is erroneous. The involvement of cervical glands may be associated with disease of lymph glands in other areas of the body, such as the mediastinum and groin. X-ray examination of the chest frequently shows widening of the mediastinal shadow in the lymphomatous tumors.

Tumors of the parotid gland. Occasionally a branchial cleft cyst will be mistaken for a tumor of the parotid gland. Because of the high incidence of branchial cleft cysts in this region (angle of the mandible), the clinician must be aware of this source of diagnostic error. Single tumors of the parotid gland can present a particularly difficult diagnostic problem. Often they are situated deep to the parotid fascia, tend to be firm, nontender, and only slightly movable. The multilobulated parotid gland tumors present no diagnostic problem. Sialography may be of some help in differentiating between these two conditions for the diagnosis of parotid gland tumor often can be established by this simple and expedient diagnostic procedure. These tumors are extremely rare in children.

Tumors of the thyroid gland. Rarely are these tumors confused with branchial cleft cysts. A tumor of the thyroid can be demonstrated to rise on swallowing.

Lipoma. These tumors may simulate branchial cleft cyst especially if deep seated. Aspiration is a valuable diagnostic aid.

Cystic hygroma. Although this tumor is encountered most commonly in infants, we have observed it in 4 adults. Of 10 cases of cystic hygromas observed on this service, 8 were found in the neck. Usually these are present at birth and tend to be soft, cystic, and nontender. Often they are multilocular and may fill the entire neck. They take origin from remnants of the jugular sac and growth is by a budding process from these rests. Cystic hygromas frequently recur. If untreated, they may extend behind the clavicle, into the axilla and behind the sternum into the mediastinum. Complete surgical removal of the tumor is mandatory if local recurrence is to be avoided.

Thyroglossal duct cyst. This cyst originates in association with the embryonic descent of the thyroid gland to its permanent position in the neck. In embryonic life, the thyroid gland is attached to the primitive pharynx by the thyroglossal duct. With the subsequent descent of the thyroid gland, this duct becomes obliterated. In 2 instances in this series, the operative diagnosis of thyroglossal duct cyst was disproved by the pathologic diagnosis of branchial cleft cyst. In each instance the operator was influenced by the fact that the lesion presented in the midline. Age is of value in differential diagnosis. Unless concomitant infection exists, the fluid may vary from transudate to exudate. It may be gray, yellow, green, or brown. Like branchial cleft cyst, the lining of the thyroglossal duct cyst may be of the squamous or of the columnar type but invariably thyroid tissue is incorporated within its wall.

Dermoid cyst. This cyst may be found in the neck. In 35 cases reviewed, 4 cases were in the neck and, in each instance, in the lateral regions of the neck. Dermoid cysts are found more commonly about the nose and the supraorbital ridge and are regarded as inclusion cysts, taking origin in the fusion of the embryonic facial processes (facial cleft theory). Dermoid cysts in the latter regions usually are deep seated, firm, nontender, and immobile but in the region of the neck, superficially placed and freely movable.

Carotid body tumors. Carotid body tumors are found rather constantly at the level of

the bifurcation of the carotid arteries. They are deep seated with small lumpy elevations on their surface and tend to pulsate with the large vessels of the neck.

STATISTICAL ANALYSIS

Distribution and occurrence. Of 90 branchial anomalies reviewed, 47 were in females and 43 were in males. This ratio of sex distribution is in keeping with the findings in the literature, for there are no references to predilection for either sex. There were 44 lesions on the right side and 39 lesions on the left side. There were 2 cases presenting directly in the midline of the neck and 1 patient complained of a large opening within the pharynx. There were only 4 instances of bilateral involvement. These data are not in agreement with those of Lahey, who stated that these lesions frequently are bilateral.

Predisposing factors. One frequently reads that hereditary influences play an important role in the etiology of these anomalies. In Virchow's case, the possibility of hereditary influence was suggested strongly. He presented a report of branchial cleft cysts occurring in a mother, her 8 children, 2 sisters and 1 brother, and 2 aunts. Review of the literature fails to support the concept of hereditary influence as an etiologic factor, nor is this concept substantiated by the data in this clinical report. In fact, on the basis of this report, we are prepared to disregard the possibility completely. In only 2 cases was trauma a precipitating factor. A small boy noticed a lump in his neck shortly after being "lassoed," and an adult male dated the onset of his cyst to a recent tooth extraction. There is little doubt that the injuries in these cases merely called attention to an already existent condition.

Age. These developmental anomalies are noticed frequently at birth and in this series of cases there were 15 such instances (16.7 per cent). Forty-one patients noted a sinus or cyst within the first year of life, thus bringing to 56 (62.2 per cent of cases) the number diagnosed within the first year of life. The average age of the patients in this group was 26½ years. The disorder was diagnosed at various stages of life, the oldest patient being 65 years of age when he first presented himself for admission (cyst). Sedgwick (12) and also Walsh (13) called attention to the fact that branchial cleft fistula is a disease of infancy whereas branchial cleft cyst is a disease found predominantly in adults. Although we are inclined to agree with Sedgwick and Walsh, it is not uncommon to examine adults with true branchial cleft sinuses or fistulas. However, infection undoubtedly plays a prominent role in such cases, the sinus resulting from an extension of the infection to the surface of the skin. This manifestation is not a common feature of branchial cleft cyst but in thyroglossal duct cyst, it is extremely common.

Infection. Of 90 patients, 39 gave the history of preceding infection such as a "head cold" or "sore throat," and examination of these patients disclosed infection of the tonsils in many instances. Quite commonly in cases of infected cyst there was associated adenopathy of the cervical lymph nodes.

Location. By far the greater number of these lesions were found close to the angle of the mandible. Neel and Pemberton, reporting on 339 anomalies of the branchial apparatus, made this same observation. In adults, this finding of branchial cyst in the region of the mandible leads frequently to the mistaken diagnosis of tumor of the parotid gland. More often than not these lesions presented along the anterior border of the sternocleidomastoid muscle. We have found them in the lateral and in the anterior portions of the neck, as well as in the midline. It is not unusual for branchial sinuses to extend upward to the postauricular regions as well as downward to the suprasternal notch and along the entire length of the clavicle. But fistulas in these rather unusual positions invariably led to the supratonsillar region of the pharynx. In short, it may be said that branchial cleft cysts are found most commonly along the anterior border of the upper third of the sternocleidomastoid muscle and that branchial cleft fistulas present an external open-

FIG. 3. The technique of excision of branchial cleft fistula through multiple incisions in stepladder fashion (after Ladd and Gross).

ing further downward in the region of the junction of the middle and lower third of the same muscle. However, other localities of the neck may be the site of these lesions.

Symptomatology. All patients gave either one or two complaints: lump or discharging sinus. Infected cysts were associated with symptoms referable to infection. Uninfected cysts were found on palpation to be oval shaped, cystic, and nontender. Usually they were palpable directly beneath the skin and were easily movable in all directions. In patients giving a history of repeated infection, the cysts were firm and only slightly, if at all, movable. The largest cyst in this series of cases measured 15 by 12 by 7 centimeters but this size was unusual, for most cysts averaged about 3 to 4 centimeters in diameter. In several cases infected cysts in the region of the mandible with accompanying regional adenopathy led to the mistaken diagnosis of cancer of the parotid gland with metastases to cervical nodes. Pain was present only in examples of cyst complicated by infection.

On presenting themselves to the hospital, 50 patients complained of only a lump, whereas 28 complained of a draining sinus, the remainder having both a lump and sinus. The latter gave the history of repeated infection with the story that the sinus opened and closed intermittently, only to be followed by further infection.

TREATMENT

If the course of the second branchial cleft is visualized as extending from a point along the anterior border of the neck of the upper pharynx, it becomes apparent that any adequate surgical procedure must have as its primary consideration the complete exploration of this entire area. Under no circumstances is it permissible to sacrifice vital structures for here we are not dealing with a malignant disease. The possibility of malignant transformation can be dismissed.

At the operating table a branchial cleft cyst may be found in one of the four following positions: (1) the cyst may be superficially located directly beneath the skin; (2) it may be of greater size and extend to the great vessels of the neck; (3) it may extend inwardly to straddle the internal and external carotid arteries and come in contact with the pharynx. This type may extend superiorly as high as the base of the brain; (4) the cyst may be a small vestigial structure situated deeply and adherent to the wall of the pharynx.

In cases of branchial cleft cyst a transverse incision directly over the mass will afford adequate exposure. Branchial cleft cyst (unlike pseudocyst with liquefaction necrosis) shells out easily. The latter type cyst is always firmly adherent to the structures within the neck and requires careful and tedious dissection.

Usually two or more transverse incisions in stepladder fashion are required to provide exposure for complete removal. However, Gross, who described the several incision technique, states that he no longer employs it. The course of these fistulas has already been mentioned. Great care must be instituted to avoid injury to the carotid artery and to the jugular vein as well as to the hypoglossal, spinal accessory, lingual, and glossopharyngeal nerves.

Exploration should always be carried to the pharynx. In branchial cleft cysts, the presence of a sinus, either exterior or inferior, is frequently absent but a fibrous strand may be present. More often than not, branchial cleft fistulas lead directly to the pharynx. Occasionally they present as sinuses to end blindly in the soft tissues of the

neck, be it the supraclavicular region, submaxillary triangle, or even at the base of the brain. The need for adequate exploration is supported by the following data. Recurrence following surgical removal of branchial anomalies occurred in 10 out of 90 patients (11.1 per cent). Quite surprisingly, 7 recurrences were in patients bearing branchial cleft cysts, whereas only 3 recurrences were in patients with branchial cleft fistulas. It is thought that recurrence following surgical removal of branchial cleft cyst is due to inadequate removal. One patient with a branchial cleft sinus underwent 5 surgical procedures before his disease finally was eradicated. The procedure of incision and drainage for infected branchial cleft cyst was carried out on 12 occasions.

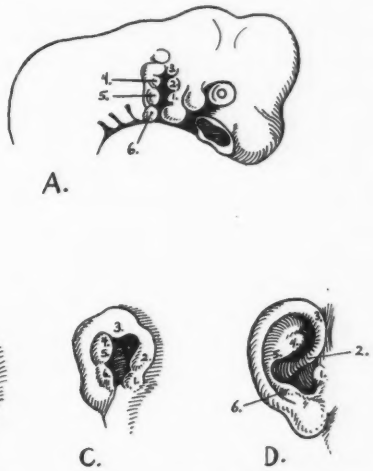

FIG. 4. The above drawings serve to illustrate the development of the external ear (after Arey). In Figure A (11 mm. embryo), elevations *1*, *2*, and *3* are seen on the first branchial arch and elevations *4*, *5*, and *6* are visualized on the second branchial arch. Examination of drawings B, C, and D reveals that elevation *1* gives rise to the tragus of the human adult ear, *2* and *3* give rise to the helix, *4* and *5* to the antihelix, and *6* to the antitragus. Preauricular sinuses originate from faulty fusion of these two branchial arches.

DISCUSSION

Most branchial cleft cysts and fistulas take origin in the second branchial cleft. This statement is supported by the fact that the usual courses of fistulas observed at the operating table correspond to the embryonic position of the second branchial cleft. Beginning at a point along the anterior border of the sternocleidomastoid muscle, a branchial cleft sinus usually proceeds anterior to the ribbon muscles, to course then along the carotid sheath. At the bifurcation of the carotid artery the sinus may terminate. This position is frequently the site of branchial cleft cyst. However, either may extend further superiorly by passing between the internal and external carotid arteries or by passing directly over the carotid sheath. Thence it proceeds under the posterior belly of the digastric muscle and styloglossus muscle, and over the hypoglossal and glossopharyngeal nerves to terminate in the supratonsillar fossa. This is the approximate course of the second branchial cleft.

Recently sinuses originating in the first branchial cleft have been reported in the literature. Rankow and Hanford presented 3 cases out of 160 defects and Byars added 3 more. Ladd and Gross were able to find but 1 case among 82 established anomalies. In the series reported herein, 4 cases were observed in which the sinus coursed directly through the parotid gland, a point suggestive that these lesions might well have originated in association with the first branchial cleft. But the history of discharge from the external auditory canal was uniformly absent. Byars and Anderson have stressed that a discharge from the external auditory canal associated with a sinus in the neck is strong presumptive evidence that the sinus originated in the first branchial cleft. The usual course of the latter sinus is as follows: From a point high up on the neck, the tract extends superiorly coursing along the anterior surface of the sternocleidomastoid muscle, digastric muscle, and external carotid artery. The tract terminates in the cartilaginous portion of the external auditory canal.

Branchial cleft cysts and sinuses often become infected and when infected cysts are examined microscopically, they may be filled with secretions and surrounded by an

unusual amount of lymphoid hyperplasia. In the presence of infection, a branchial cleft cyst with its lymphoid tissue assumes the role of a regional lymph node. Consequently, the associated lymphadenitis is responsible for varying amounts of fluid. Palpation at this time reveals a tender cystic mass. With increasing frequency of infection, the cyst becomes progressively firmer. In the noninfected cyst, the wall of the cyst usually measures 1 millimeter in diameter. However, in a cyst which has undergone prolonged infection, its wall may measure 1 centimeter in diameter. Often, after repeated infection, the lining may be replaced by keratin. When the latter change occurs in a branchial cyst, it cannot be distinguished from the ordinary inclusion cyst.

To conclude this discussion we wish to mention a word concerning malignant tumors arising from branchial anomalies. Lahey has stated that not a few of the movable tumors of the neck, such as branchial cysts, become malignant. Such has not been our experience nor does this concept receive support from a review of the literature. We have observed 2 cases of branchial adenomas and 1 of carcinoma which might questionably have originated from the branchial apparatus. It is our feeling that earlier references to carcinomas originating from lesions of the branchial apparatus probably applied to primary carcinomas of the thyroid gland or tumors metastatic to the neck.

SUMMARY

1. Ninety cases of branchial cleft anomalies have been reviewed and their clinical features analyzed.

2. Sixty-two per cent of all branchial anomalies were diagnosed within the first year of life.

3. The embryology of the branchial apparatus has been reviewed and particular attention has been accorded to the numerous possibilities for faulty anatomic development.

4. The pathogenesis of branchial cleft cysts and fistulas is discussed.

5. The recurrence rate following surgical removal of branchial anomalies was 11.1 per cent.

6. The earlier teaching that malignant tumors originate frequently in branchial anomalies is not substantiated by this review of cases.

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RECENT EXPERIENCES WITH PROSTATIC ABSCESS

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MANY LARGE SERIES of prostatic abscesses were reported and the subject was discussed frequently prior to the introduction and widespread use of antibiotics. The reported mortality ranged from 6 to 30 per cent. A contributing factor to the relatively high incidence of the disease in the past was the ineffectiveness of the treatment of gonorrhea, with the resulting development of prostatic abscess. The incidence decreased sharply with the advent of sulfonamides and antibiotics. This is reflected in the appearance of isolated case reports rather than larger series. The clinical entity frequently is not considered in the differential diagnosis of lower urinary tract diseases.

There have been 12 cases of prostatic abscess in the University Hospitals of Cleveland since 1944. Each of these was proved by operation or urologic investigation. This study does not include patients in whom a prostatic abscess was suspected, but in whom spontaneous recovery occurred and positive findings were not sufficient to establish a diagnosis. There were 9 such cases during this period. Ages of the 12 patients with proved prostatic abscesses ranged from 30 to 80 years. Four patients were under the age of 50. Gram stains and cultures of urine and abscess material were obtained in almost every case. Blood cultures and antibiotic sensitivity studies were performed when indicated.

Eight of the 12 proved cases have occurred in the last 2 years. This comparatively increased incidence and the recent lack of emphasis of this disease process as a clinical entity stimulated the present report. The difficulty of establishing a diagnosis and the appreciable morbidity and mortality of this disease further served as indications for this study.

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RESULTS

A summary of the presenting symptoms and signs, admission diagnoses, and operative treatments of the 12 patients in this series is tabulated in Table I. The only death occurred in a 50 year old negro (Case 2) whose history will be summarized briefly because it is representative of the majority of patients in this series.

This patient entered with the chief complaint of dysuria, urgency, frequency, and low back pain of 3 days' duration. The past history revealed that for many years he had had diabetes mellitus, which was controlled by 40 to 50 units of 2:1 insulin. His temperature was 39.5 degrees C. at the time of admission to the hospital. The patient was a thin, poorly developed male who was obviously acutely ill. There was a harsh systolic aortic murmur. Rectal examination revealed the prostate gland to be twice as large as normal, boggy, and tender on palpation of the right lobe. Urinalysis revealed many white blood cells in the voided sediment. The peripheral white blood cell count was 15,200 per cubic milliliter. The patient was given 300,000 units of aqueous procaine penicillin and 2 grams of streptomycin intramuscularly every day. He had a moderately febrile course for the first 72 hours and then developed shaking chills and a temperature of 39.9 degrees C. Rectal examination now revealed fluctuation throughout the region of the right lateral lobe of the prostate gland. Incision and drainage of a large abscess that completely replaced the prostate gland was performed, and intensive chemotherapeutic and antibiotic treatment was continued. Despite active supportive therapy, the patient continued to have a high fever and to show signs of uncontrolled infection. He expired 11 days postoperatively. Several days prior to death digital examination of the perineal wound failed to reveal any residual collection of pus. A coagulase positive staphylococcus aureus was isolated repeatedly from urine and blood cultures prior to death. Autopsy permission was not granted.

TABLE I.—SUMMARY OF CASES

No.	*Age*	*Diabetes mellitus*	*Presenting symptoms*	*Signs*	*Original diagnosis*	*Duration of symptoms prior to admission*	*Hospital course*
1 U.H. 631–921	42	No	Retention, dysuria, perineal pain, penile pain	Prostate 3x enlarged, tender, boggy, tense in left lobe	Acute prostatitis, stricture	4 days	Incision and drainage of prostatic abscess, recovered, discharged 12 days after surgery
2 U.H. 154–341	56	Yes	Urgency, frequency, dysuria	Prostate 2x enlarged, boggy, tender, not tense	Acute prostatitis, ? abscess	3 days	Incision and drainage of prostatic abscess, Died 11 days after surgery
3 U.H. 208–868	40	Yes	Chills, fever, retention, perineal pain	Perineum tender, prostate 3x enlarged, tender and tense	Acute prostatitis, stricture, ? abscess	7 days	Spontaneous rupture into urethra and perineum; incision and drainage of perineal abscess, recovered, discharged 21 days after surgery
4 U.H. 288–224	57	No	Chills, fever, dysuria	Periprostatic & prostatic tenderness	Rectal abscess	2 wks.	Extension to periprostatic tissue; incision and drainage of prostatic abscess, recovered, discharged 8 days after surgery
5 U.H. 239–076	53	No	Chills, fever, dysuria, retention	Prostate enlarged 1x. Crater in rt. lobe	Acute prostatitis	2½ wks.	Spontaneous rupture into urethra. Recovered, discharged 2 wks. after admission
6 U.H. 639–309	30	No	Dysuria, frequency, perineal pain, penile pain	Prostate 2x enlarged, tender, boggy, fluctuant	Gonococcal urethroprostatitis	10 days	Incision and drainage of prostatic abscess, recovered, discharged 9 days after surgery
7 U.H. 245–129	50	No	Frequency, dysuria, retention	Prostate enlarged 2x. Rt. lobe tense and tender	Acute prostatitis	2½ wks.	Incision and drainage of prostatic abscess, recovered, discharged 6 days after surgery
8 U.H. 658–177	36	Yes	Perineal pain	Prostate tender, fluctuant	Acute prostatitis, abscess	2 wks.	Incision and drainage of prostatic abscess. Recovered, discharged in 2 wks.
9 U.H. 277–237	66	Yes	Retention, frequency	Prostate enlarged 3-4x	Benign prostatic hypertrophy	3 days	At exploration for removal of benign enlargement, a prostatic abscess was found and drained. Pt. recovered, discharged 21 days after surgery
10 U.H. 646–309	30	No	Rectal pain, retention	Prostatic & periprostatic tenderness	Stricture, rectal abscess	2 wks.	Cystotomy; incision and drainage of prostatic abscess. Recovered, discharged 2 wks. after admission
11 U.H. 607–012	50	Yes	Hematuria, dribbling, retention	Enlarged 2–3x, slight tenderness at base	Benign prostatic hypertrophy	Many wks. 3 days of acute symptoms	Incision and drainage of prostatic abscess. Recovered, discharged 2 wks. after surgery
12 U.H. 621–091	69	Yes	Frequency, dysuria, dribbling	Prostate enlarged 2-3x, exquisitely tender	Acute prostatitis	2 wks.	Chemotherapy for 10 days. Incision and drainage of prostatic abscess. Discharged 4 wks. after admission

As recorded in Table I, retention of urine and pain with attempted urination were the most common presenting symptoms. Four of the patients (Cases 1, 2, 9, and 11) had clinical manifestations for less than 1 week prior to admission. There was periprostatic extension at the time of surgical treatment in 2 patients (Cases 3 and 4). Surgical treatment was delayed because there was difficulty in making the diagnosis in 1 of these patients (Case 4), and there was delay by the patient in seeking medical aid in the other instance (Case 3). Marked relief of pain and rapid decline in temperature occurred following drainage in all patients who survived.

A summary of the bacteriologic studies and antibacterial treatments is shown in Table II. It can be seen that all patients received antibacterial therapy prior to the time a diagnosis of prostatic abscess was established, because lower urinary tract inflammatory disease was thought to be present. Gram positive cocci were cultured in the majority of the cases. Positive cultures of urine or abscess material were obtained in every instance except 2; in 1 of these cases, gram positive cocci were demonstrated in a smear of the abscess material.

DISCUSSION

Abscess of the prostate gland occurs most frequently following posterior urethritis and acute prostatitis. The differentiation between acute, diffuse, nonsuppurative prostatitis and abscess formation may be extremely difficult because urgency, frequency, and dysuria are the usual symptoms in both processes. The early administration of antibacterial agents may serve to mask systemic symptoms and add to the difficulty in diagnosis. Also, a tense, exquisitely tender gland may disguise the presence of an abscess and demonstrable fluctuation may not be encountered. In 4 instances the onset of shaking chills and a rise in temperature were the first evidences of the presence of an abscess. A brief period of illness similarly does not rule out the possibility of abscess formation having occurred, as evidenced by the number of our patients in whom symptoms were present for less than 7 days.

A history of unusually severe and persistent perineal and rectal pain, occasionally associated with rectal urgency, should immediately lead to the suspicion of a prostatic abscess. Pain, without associated urinary symptoms, may be the chief complaint, as illustrated in the following case report (Case 8).

A 33 year old male was admitted to the hospital with the chief complaint of severe pain in the perineum of 2 days' duration. Two weeks prior to admission he had noted an intermittent perineal discomfort associated with coughing, sneezing, and bowel movements. There were no urinary symptoms. Twelve hours prior to admission the patient had a shaking chill. He was known to have had diabetes mellitus which had been controlled by diet for 1 year. The patient was a well developed, well nourished male in acute distress. Temperature was 39.2 degrees C. and pulse was 128. Urinalysis revealed 40 to 45 white blood cells per high power field and no acetone or sugar. Physical examination was essentially negative except that the prostate gland was slightly enlarged, moderately tender, and definitely fluctuant in the midline. Incision and drainage by the perineal route was done immediately. There was a large abscess cavity that completely replaced the prostate gland. The patient did well postoperatively and he was discharged 2 weeks after admission.

The location and nature of the pain may lead to confusion with perirectal inflammation. Usually the pain of prostatic abscess is severe and unremitting. In 2 cases (Cases 1 and 6), the pain radiated down the penis.

The age of the patient should be no deterrent to a consideration of the diagnosis. The majority of our patients were in the age group in which hypertrophy and neoplastic enlargement of the prostate normally occur. In 6 cases the patient was admitted because of urinary retention; a tentative diagnosis of benign prostatic hypertrophy was made in 2 of these instances (Cases 9 and 11). The following summary illustrates the course of 1 of these patients.

A 50 year old white male was admitted in acute retention. History was somewhat uncertain because of language difficulties. Apparently, he had had hesi-

TABLE II.—SUMMARY OF BACTERIOLOGIC STUDIES

		Cultures	*Gram stain of abscess material*	*Before definitive diagnosis*	*After definitive diagnosis*
1	Urine	Alpha hemolytic strep.	Gram positive cocci	Gantrisin	Penicillin
	Blood	No growth		Penicillin	Streptomycin
	Abscess	No growth		Streptomycin	
2	Urine	Staph. aureus	Gram positive cocci	Terramycin	Gantrisin
	Blood	Staph. aureus		Penicillin	Penicillin
	Abscess	Staph. aureus, E. coli		Streptomycin	Streptomycin, terramycin
3	Urine	Proteus vulgaris		Penicillin	Penicillin
	Blood	No growth		Gantrisin	Streptomycin
	Abscess	No growth			Gantrisin
4	Urine	No growth	Gram positive cocci	Penicillin	Penicillin
	Blood	No growth			Sulfadiazine
	Abscess	No growth			
5	Urine	No growth		Sulfathiazole	Sulfathiazole
	Blood	Staph. albus			
	Abscess				
6	Urine	Staph. aureus	Gram positive cocci	Penicillin	Penicillin
	Blood	Alpha hemolytic strep., Beta hemolytic strep.		Streptomycin	Streptomycin
	Abscess	No growth			
7	Urine	Staph. albus	Gram positive cocci	Sulfathiazole	Sulfathiazole
	Blood	No growth			Sulfadiazine
	Abscess	Staph. aureus			
8	Urine	E. coli	Gram positive cocci	Achromycin	Penicillin
	Blood	E. coli			Streptomycin
	Abscess	No growth			Achromycin
9	Urine	E. coli	Gram positive cocci	Gantrisin	Penicillin
	Blood	No growth			Streptomycin
	Abscess	E. coli			
10	Urine	Specimens lost			Penicillin
	Blood				Streptomycin
	Abscess				
11	Urine	Aerobacter aerogenes Nonhemolytic strep.		Gantrisin	Penicillin
	Blood	No growth			
	Abscess	Staph. aureus			
12	Urine	E. coli, Micro. tetragenes	No organisms seen	Penicillin	Terramycin
	Blood	No growth		Streptomycin	
	Abscess	E. coli			

tancy, urgency, frequency, and nocturia for several years. Three days prior to admission these symptoms became more severe and he developed dribbling and overflow incontinence. He was known to have been a mild diabetic for many years. On admission he was found to have a distended bladder. The prostate gland was enlarged 3 times greater than normal, the median sulcus was obliterated, and there was a small tender area at the left base. Urinalysis showed only an occasional white blood cell per high power field and a 2+ Benedict's reaction for sugar. Peripheral white blood cell count was 10,000. The patient was studied for the first few days following admission. Repeat rectal examination on the fourth hospital day showed increased tenderness and fluctuation on the left side of the prostate gland. Perineal incision and drainage was performed. The patient did well postoperatively and was discharged after 12 days.

The known tendency of patients with diabetes mellitus to develop infections should raise one's index of suspicion when lower urinary tract symptoms develop in a diabetic person. Six patients in this series had diabetes mellitus; the only fatality was in a diabetic patient. Seemingly, the best course to pursue would be immediate hospitalization and close observation of diabetic patients with acute prostatitis. Although it has been stated that prostatic abscesses are usu-

ally metastatic, this mechanism was not evident in any of the patients in the present series.

Despite continued efforts to obtain more effective urinary tract antiseptics, the increasing incidence of resistance of organisms to these agents is well known. Ineffectiveness of antibiotics in the treatment of chronic prostatitis has been reported previously by the authors.[1] The failure of antibacterial drugs to effect the resolution of a frank abscess has been repeatedly observed. These factors are probably responsible for the apparent increasing incidence of prostatic abscess despite increased effectiveness of the drugs utilized in the therapy of gonorrhea in recent years. There was a history of an antecedent gonococcal infection in only 1 patient in this series (Case 6).

Gram positive cocci were the predominant group of pathogenic organisms in this series. Their presence as the major etiologic bacteria is believed to be the result of the increasing incidence of development of resistant strains of staphylococci to penicillin, and the susceptibility of other organisms to different antibiotics. Since the incidence of cross resistance of penicillin resistant staphylococci to other antibiotics has been reported as high as 70 per cent by Fusilla,[2] it seems unlikely that utilization of broad spectrum antibiotics will alter the necessity for surgery. The variety of organisms that may be cultured at the time of inflammatory diseases of the prostate gland is a further indication that suppuration will continue to develop despite the liberal use of drugs.

Perineal aspiration or transurethral prostatotomy have been suggested by Timberlake[3] as aids to the earlier diagnosis of fluctuation when a tender, swollen prostate gland is found. We believe that perineal incision should be employed earlier when the diagnosis is suspected. Early drainage is followed by immediate relief of pain and it prevents extracapsular contiguous extension of the process.

CONCLUSION

Abscess of the prostate gland should be suspected in patients, particularly diabetics, who have inflammatory diseases of the lower genitourinary tract, when the disease is accompanied by severe perineorectal pain and a tender, enlarged prostate gland. Diagnosis and definitive therapy should not await the development of local fluctuation as palpated per rectum. The continued administration of antibacterial drugs in the presence of an actual abscess is of little value and it may mask the systemic manifestations of spread of the infection and thereby delay definitive surgical treatment. Once the diagnosis is made, immediate incision and drainage, preferably by the perineal route, should be performed.

[1] SCHATTEN, W. E., and PERSKY, L. Aureomycin therapy of chronic prostatitis. Surg. Gyn. Obst., 1954, 98: 40–44.

[2] FUSILLA, M. H., ROMANSKY, M., and KUHNS, O. M. Further investigation on cross resistance as well as studies on induced sensitivity to antibiotics. Antibiot. Chemo Ther., 1953, 3: 35–40.

[3] TIMBERLAKE, G. An electro-prostatome; relief of prostatic abscesses and acute obstructive prostatitis by transurethral prostatectomy. J. Urol., Balt., 1938, 40: 343–345.

EDITORIALS

SURGERY OF THE HIP IN ADULTS

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SINCE THE SMITH-PETERSEN NAIL and later the vitallium mold were introduced by Smith-Petersen, surgical procedures for various affections of the hip, particularly in adults, have had widespread acceptance. Many types of operative procedures have been devised and many devices have been developed, particularly various prosthetic replacements for the femoral head.

The wide variety of these and their adoption by many surgeons suggest that the results of surgical procedures cannot be altogether satisfactory or so many different types of prostheses would not be developed. That most of the lesions of the hip for which surgical procedures have been devised are severely disabling must be recognized. That most of the surgical procedures have given considerable relief must be recognized also. That enthusiasm for surgical treatment of disabilities of the hip may carry use of surgical measures beyond the limits of usefulness is a danger not to be overlooked. Experience should teach physicians and surgeons alike that all patients are not suitable for one type of operation but that careful selection and preparation of the patient for operation are as important as the operative procedure itself and also that a patient's best interests often may be served by advising against an operation.

The function of the hip may be considerably impaired and yet the patient may not be seriously handicapped. Indeed, complete loss of motion of the hip may be only moderately disabling provided that ankylosis is in a good position for easy ambulation. Patients often learn to accommodate themselves to a stiff hip not only when standing and walking but even when sitting.

Pain and deformity, then, are the main symptoms which bring the patient to the point of seeking medical care and advice. Many patients with mild or moderately severe pain only after exertion can be treated conservatively with at least temporarily beneficial results. Important points in such a program are: reduction in weight, which is so often needed, heat and massage to improve the local conditions about the joint, and limitation of certain activities, particularly those which may require extremes of motion, such as squatting and horseback riding. Often use of a cane may throw the balance away from the painful hip enough to minimize the pain.

However, when the pain has been of long standing, when deformity has developed, and when roentgenograms show marked thinning of the joint space with degenerative and hypertrophic changes, operation is indicated. Before it is undertaken the patient should be thoroughly informed as to what is involved. This should include information concerning the period of hospitalization, the period after operation that crutches may be necessary, and the long and tiresome program of exercise which the patient must follow if the best result is to be obtained. Patients who are unwilling to undergo a

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strict reduction program when obviously overweight or who are unwilling to cooperate in the long program of postoperative rehabilitation are probably best left alone and operation should not be advised.

When operation is decided on, the choice of operation may best be left to the judgment of the surgeon at the time of operation. When the joint is exposed, he may find conditions which require variations in the plan for the operative proceedings. It is well to inform the patient before any operation of the possibilities. Thus he will not be surprised or disappointed because the operation performed was different from what he had expected.

This is not the place for a detailed discussion of technical details of surgical procedures. Assuming that a good job is done and the operation is carried out to the satisfaction of the surgeon, much remains to be done in order to obtain the best result. The primary postoperative care is important but as soon as the wound has healed a program of exercise designed to restore muscle strength to as near normal as possible must be instituted. No more important part of the therapy exists than this phase of the treatment. Failure to get the maximal result comes frequently because the patient fails to appreciate the importance of this aspect of the treatment and, either through misunderstanding or unwillingness, fails to work diligently on this program. In some instances an uncooperative attitude may be due to the surgeon's failure to emphasize the importance of the program to the patient.

Patient, careful work must be carried out with detailed attention to the exact type of exercise. The muscles about the hip have often either not been used or have been used abnormally for months. To bring them back to normal function requires the utmost in effort on the part of the patient, the physiotherapist, and the surgeon. In some instances, when the lesion of the hip has been of relatively short duration, the strength and function of the muscles may be regained rapidly. Until such restoration is complete, the best results will not be obtained from arthroplasties on the hip, for the stability and normal function of the hip are dependent on normal musculature, and if the patient is to walk without a limp restoration of muscular strength and function is necessary.

Although the idea of immediate replacement of the femoral head with a prosthesis in cases of acute subcapitular fractures of the neck of the femur has been widely accepted, it has not been accepted by all. There is no doubt that the argument in favor of such a procedure for elderly and debilitated patients is a strong one because such patients are not suitable candidates for the long period of treatment that may be necessary when hip nailing is done. Careful selection of cases may justify this procedure, but efforts to restore union without a prosthesis should be carried out for the average fracture of the hip when the patient's general condition offers no contraindication to the procedure.

Cup arthroplasty and prosthetic replacement of the head of the femur will fail in a certain percentage of cases in which they are employed. Such failures may be due to infection, which in nearly all cases requires removal of the cup or prosthesis and some type of further operative procedure. In some cases arthrodesis may be possible and can be performed. In a good many instances, however, a successful arthrodesis is difficult to accomplish. In a number of such cases the author has resorted to the Colonna type of reconstruction operation with satisfactory results. Lewis and the author[1] have recently reported a follow-up study on such a series. In most of these cases infection can be controlled, and a stable hip and relief from pain can be obtained with the Colonna operation. The range of motion of the hip is often limited but, compared to arthrodesis, most patients are much better satisfied to have at

[1]Lewis, R. C., and Ghormley, R. K. Colonna reconstruction of the hip: results in 57 cases. Proc. Mayo Clin., 1954, 29: 605–613.

least a partial range of motion, provided they are free of pain and have a leg on which they can bear weight.

As time passes and more experience with surgical procedures on the hips is obtained, good results will be seen more uniformly. Much has already been accomplished in relieving many patients of severely disabling hip conditions and restoring many hips to a reasonable degree of function. Many patients expect too much in the way of restoration, and the surgeon should advise the patient that in most instances a normal hip will not be obtained. Some hips are nearly normal, it is true, and many of the patients are able to do all the things that a person with normal hips will do. Careful selection of cases and careful planning by the surgeon for the operation, and the program of aftercare, with the patient fully understanding his part in the program, will produce more uniformly good results.

THE DUODENAL STUMP

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SUBTOTAL GASTRECTOMY for duodenal ulcer has become, during the past two decades, a relatively safe operation. Improvements in anesthesia, in the nutritional preparation of patients and advancements in knowledge concerning the use and abuse of gastric suction and intravenous fluids have all played an important part in reducing the hazard of this valuable and common operation. Two main difficulties, pulmonary embolism and peritonitis from a leaking duodenal stump, remain to account for the vast majority of the postoperative deaths.

It seems that, in our present state of knowledge, little more can be done to reduce the incidence of the former. Being quite ignorant of the cause of venous thrombosis in most cases and, indeed, in most fatal cases being unaware of its presence until the awful moment of the lodgment of the embolus, the surgeon is usually helpless to prevent it. He is, perhaps, justified in regarding such an event as unavoidable. This is not the case when death is caused by peritonitis from a breakdown of the sutured duodenal stump. Here the operator must accept full responsibility for it is evident that there has been an error in judgment or technique at the time of operation, an error that he might, with greater caution, have avoided. This is not to say that a surgeon can always find a way out of the difficulty or, to put it another way, that he can invariably avoid finding himself in a position where he has passed the "point of no return" and is faced with the problem of closing a short, tattered duodenal stump. There must be very few who have such consistent prescience, confidence, and skill that they do not occasionally find themselves in this dilemma. Experience and dexterity will usually provide the answer but it is probable that even in the best of hands the closure of the stump will, at times, be precarious.

Recent articles have raised the possibility that there may be an easy way out. To anticipate a breakdown of the suture line and to insert a catheter into the partially closed duodenum at the time of operation is an attractive proposition. In the reported cases this procedure has been attended by satisfactory results to an extent that the credulous surgeon might feel that his troubles in this respect are now overcome. That this may not be the case is a point that should be discussed. Certain difficulties may still arise and have, actually, been observed. In the first place, the tube may not function at all, due to obstruction of its lumen, kinking, or to the fact that it may come out of the duo-

denum. These are all mechanical faults due to imperfect technique but they do happen and the situation is not easily rectified.

Secondly, though the tube may function, there may be leakage of duodenal and pancreatic fluids around it which may not all find its way out along the Penrose drains that are usually placed alongside the catheter, and in some cases a diffuse biliary peritonitis or subhepatic or subphrenic abscess may develop.

Thirdly, although a firm fistulous tract may form and there may be no leakage at any time, the fistula may persist. Cases have been reported in which the fistula persisted for many months and operative excision of the fistula was required for cure. This complication perhaps may not be regarded as a serious one and may be accepted as a small price to pay.

Finally, there may be such profuse drainage through the catheter that great difficulty may be encountered in maintaining fluid balance. This is likely to occur if there is stomal obstruction, but may come about even when there is no such lesion. Welch[1] has met this problem by performing a jejunostomy through which he returns the duodenal drainage and this should usually be effective.

This technique might not, however, overcome a complication that was encountered recently, but that has been hitherto unreported. The patient illustrating this complication was a young man with a large posterior wall duodenal ulcer that extended down to within a few millimeters of the common bile duct. The duodenal stump was closed about a catheter which was brought out through a stab incision. For a time the catheter drained a small amount of duodenal juice and there was no leakage, but after a few days there was leakage about the catheter and a sump drain was inserted along the catheter. Through this drain and through the catheter there poured a tremendous amount of duodenal content. On the eleventh postoperative day the patient passed a small amount of blood in his stool and from that time on his course was one of almost continuous gastrointestinal hemorrhage of which he died on the fifteenth postoperative day. Autopsy showed a subphrenic abscess on the right side and a massive jejunal ulcer on the efferent loop just below the anastomosis. It was thought that probably this jejunal ulcer resulted from the unopposed action of the gastric juice resulting from the fact that all his duodenal and pancreatic secretions were passing through the fistula. Thus, the possibility of jejunal ulceration may have to be added to the list of potential complications of duodenostomy.

There is no doubt whatever that catheter drainage of the duodenal stump is the safest way out when firm closure of the stump cannot be achieved. The incidence of complications and the mortality in these difficult cases will almost certainly be reduced if this method is used. Sufficient experience with it has now been gained, however, to warrant the assertion that the technique is by no means foolproof, that serious complications can and do occur, and that its use should be reserved for those cases in which all reasonable means of attaining safe closure have been exhausted rather than as a routine in all cases in which at first it appears that the stump will be hard to close.

[1]Welch, C. E., and Rodkey, G. V. A method of management of the duodenal stump after gastrectomy. Surg. Gyn. Obst., 1954, 98: 376–379.

CONSTRUCTION OF A SACROCERVICAL LIGAMENT FOR UTERINE SUSPENSION

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VARIOUS OPERATIVE TECHNIQUES for the correction of retrodisplacement of the uterus have their proponents and opponents, and there are those who would place all such operations in the category of unnecessary surgery. This difference of opinion is not surprising when it is realized that the operations currently in use for uterine suspension, such as shortening the round ligament or shortening the uterosacral ligaments, have distinct limitations in the presence of structural inadequacy of the uterine supports.

A method of supporting the cervix and better controlling the mobility of the uterus by constructing a sacrocervical ligament and making it entirely retroperitoneal is presented herein. The method has been used with very favorable results in 22 consecutive cases in the past 4 years and appears to obviate the possibility of failure in those cases in which the ligaments are structurally inadequate to support the cervix and prevent a recurrence of uterine descensus and prolapse.

Retrodisplacement is not merely a tipping backward of the uterus with the cervix acting as the pivotal point, but rather involves several mechanical factors. First, the pelvic axis is somewhere near the middle of the uterus and the mechanism of retrodisplacement is rotation around this axis and descent of the pivotal point, with the cervix swinging forward and the fundus of the uterus backward. Second, and as a consequence, there is a lengthening of the uterosacral ligaments, followed by dropping of the cervix in the vaginal canal, causing a shortening of the anterior vaginal wall. Third, the round and broad ligaments also lengthen because of atony or stretching.

Factors that assist in retaining the uterus within the normal limits of its mobility "or assist in preventing its remaining permanently beyond the normal limits," are, according to Crossen: "(1) The pelvic floor; (2) the sacrouterine ligaments; (3) the broad ligaments; (4) the round ligaments; (5) the normal weight and size of the uterus; and (6) the normal tone and fullness of the pelvic tissues."

As Wharton (3) has stated, so long as the broad and the uterosacral ligaments are strong, the cervix maintains its normal position in the pelvis. "If, however, these ligaments lose their tonicity and strength, the weight of the uterus, plus intra-abdominal pressure, forces the cervix to continually lower levels in the pelvis. This is the mechanism by which uterine descensus develops; it is frequently accompanied by retrodisplacement." Again, "The uterosacral ligaments are the most variable of all; they may be either slender folds of peritoneum or strong fibrous bands. When they are strong and taut, they hold the cervix in the posterior part of the pelvis, and prevent uterine descensus and prolapse" (4).

It has been stated by Davis that most gynecologic surgeons are convinced that shortening of the uterosacral ligaments increases materially the percentage of satisfactory results which follow suspension operations. Obviously, however, if the ligaments that are used to suspend the uterus are not strong enough to hold it, the operation will fail. "This general principle," Wharton (5) contends, "is the basis of choice between the available operations."

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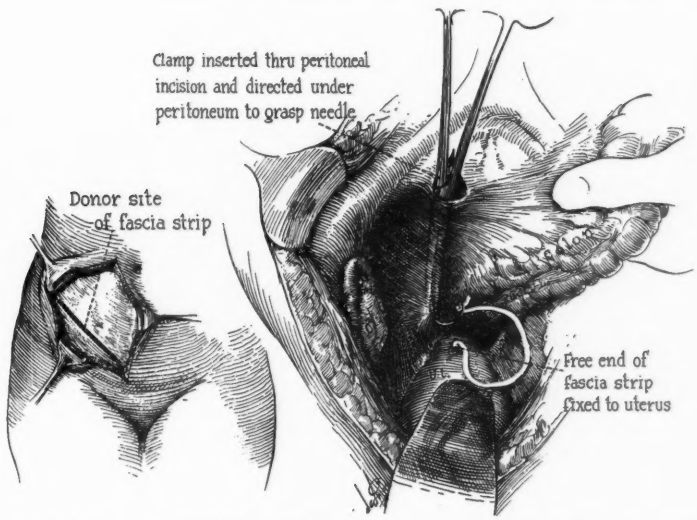
Clamp inserted thru peritoneal incision and directed under peritoneum to grasp needle
Donor site of fascia strip
Free end of fascia strip fixed to uterus

FIG. 1. Left, The fascial ligament is taken from the aponeurosis of the external oblique muscle. Right, This fascial strip is attached to the cervix at the junction of the uterosacral ligaments and grasped by a Kelly clamp to be drawn retroperitoneally.

These observations by authorities, and personal experience over the last 25 years, have led to the opinion that failures encountered in the treatment of retrodisplacements are due to a poorly chosen operation for the individual case, to technical failure, or to anatomic inadequacy of the structures. It was in an attempt to correct the third factor that the following operative procedure was devised. In this small series of cases it has proved to be efficacious in correcting structural inadequacy—one of the causes of retrodisplacement.

PROCEDURE

The abdomen is entered through a Pfannenstiel or right paramedian incision, and the pelvis is examined for pathologic changes and for mobility of the uterus. Ordinarily, the cervix can be moved a distance of from 2 to 4 inches, indicating great relaxation of the broad, round, and uterosacral ligaments. The skin, superficial fascia, and adipose tissue are dissected away from the external oblique fascia and retracted toward the right groin. A strip of the external oblique fascia, ¼ inch by 5 inches, is excised 1 inch from and parallel with the inguinal ligament (Fig. 1, left). This is threaded through a small Gallie fascia suture needle and used to construct a new uterosacral ligament. The anterior rectus sheath cut longitudinally is not suitable for this because the obliquity of its component fibers causes it to separate when under tension. The defect in the external oblique fascia is closed with a running suture of chromic catgut or interrupted cotton sutures.

The rectum and sigmoid colon are retracted to the left and the posterior peritoneum is elevated with forceps and incised longitudinally over the promontory of the sacrum, distal to the bifurcation of the aorta. This incision need be only 1 inch long but care must be taken not to injure the middle sacral artery, the ureter, or the presacral plexus of nerves. A Kelly clamp is inserted retroperitoneally, toward the cervix, so that the point of the clamp is kept in close apposition to the peritoneum (Fig. 1, right).

The needle, threaded with fascia, is passed through the cervix, 5 millimeters deep, at the junction of the uterosacral ligaments and the cervix, and the distal end of the fascia is secured with 1 cotton suture. The needle is next passed through the elevated peritoneum of the cul-de-sac and is grasped by the Kelly clamp to be drawn out retroperitoneally through the peritoneal incision over the promontory of the sacrum (Fig. 2A). The needle is then passed

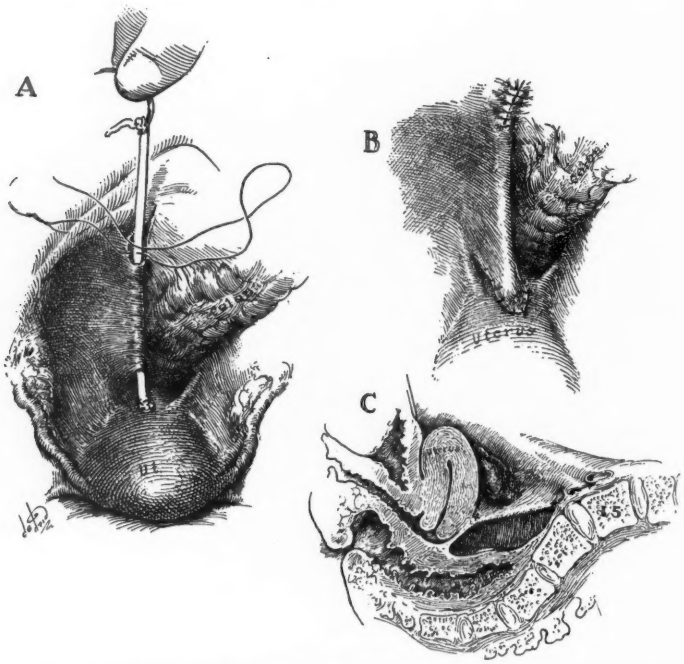

FIG. 2. A, The new ligament is attached to the periosteum over the promontory of the sacrum after elevating the cervix to the desired position. B, The new ligament is covered by the peritoneum of the cul-de-sac and the presacral peritoneum is closed, making the entire ligament retroperitoneal. C, The corrected position of the uterus is maintained by the newly constructed sacrocervical ligament.

through the periosteum on the anterior surface of the sacrum, the fascial "ligament" is drawn taut to elevate the cervix, and the proximal end is secured with 1 cotton suture.

The excess fascia attached to the needle is cut away to be discarded and the posterior peritoneum is closed with a running suture of No. 00 chromic catgut on an atraumatic needle (Fig. 2B). The peritoneum of the cul-de-sac is then pulled up around the new sacrocervical ligamentous attachment and sutured in place to make this new ligament entirely retroperitoneal. Figure 2C shows the corrected position of the uterus after construction of the new ligament (lateral view).

This procedure elevates the cervix and throws the uterus forward, as can be observed even while the patient is on the operating table. A Baldy-Webster round ligament plication and closure of the abdomen may then be done in the conventional manner. In the most recent cases the Baldy-Webster procedure has been omitted, with equally good results.

Between February 1951 and February 1955, 22 consecutive patients with retrodisplacement of the uterus have been operated upon by the procedure described or in a similar manner.

RESULTS

The original operation was an attempt to form a ligament by means of passing a linen suture retroperitoneally from the cervix to the sacrum. In only 1 case was this done. The cut edge of rectus sheath was next used because of its ready availability when operating through a longitudinal incision. However, because of the oblique direction of the fibers of the rectus muscle sheath, when a longitudinal strand was cut from it, the strand tended to separate as it was drawn through the sacral periosteum. This necessitated suturing the proximal end of the newly formed ligament to the periosteum with cotton instead of passing the

ligament through a bite of periosteum and resulted in a less durable attachment. Nine patients were treated in this manner before the method was abandoned and a source of stronger material searched for.

In 3 cases dermal graft ligaments were made from excised abdominal scars, the cornified epithelium being removed by a scalpel or scissors and the ligament attached as previously described. This forms an excellent ligament and tends to shorten as fibrosis of the cutis occurs. In the last 9 cases in the series the ligament was derived from the external oblique aponeurosis as shown in Figure 1 and was found to be most satisfactory.

Antibiotics were employed in only the 3 cases in which dermal grafts were used to construct the sacrocervical ligament. In no instance has infection or other complication occurred. Twenty-one of the 22 patients had additional surgery such as an appendectomy, colporrhaphy, cauterization of the cervix, myomectomy, or resection of areas of endometriosis. The patients ranged in age from 18 to 44 years, the average age being 32.7 years. In the 2 patients with endometriosis in whom sacrocervical suspension was done, the endometrial implants could be resected. The procedure has not been used in cases of advanced endometriosis because of the danger of retroperitoneal inoculation with endometrial cells.

Because of incapacitating dysmenorrhea 2 patients received presacral nerve resections in addition to a combined Baldy-Webster and sacrocervical ligament construction. Both patients are now well; 1 is pregnant.

After completing 17 operations combining the Baldy-Webster procedure with construction of a sacrocervical ligament, and observing the results, it was deemed safe to omit the shortening of the round ligament. It had long been my contention that a sufficiently strong sacrocervical ligament was adequate to support the uterus in patients who had retroversion with prolapse and to prevent a recurrence. Accordingly, the last 5 patients in the series were treated by means of ligament construction only. It has been gratifying to observe that this sacrocervical ligament support alone raises the cervix in the pelvis and holds the uterus in an anteverted position even while the patient is on her back on the operating table.

The results to the present have been consistently good. In no instance in the entire series has sacrocervical suspension failed to hold the uterus in an anteverted position whether it was done alone or combined with round ligament shortening. One patient who received the combined operation is now pregnant, which will put the procedure to the supreme test of its efficacy.

SUMMARY

A method of surgical treatment of retrodisplacement of the uterus is described in which a sacrocervical ligament, entirely retroperitoneal, is constructed.

The procedure was designed to correct one of the major causes of retrodisplacement of the uterus—anatomic inadequacy of the supporting structures.

The procedure has been used in 22 cases from February 1951 to February 1955. Results were uniformly good as far as maintaining the uterus in proper position was concerned. In 2 instances dysmenorrhea persisted, although in lesser degree, but there have been no other complications, no instances of infection, and so far there has been no evidence of weakening of the ligamentous support.

The last 5 patients in the series were treated by ligament construction only, omitting the additional step of shortening the round ligaments. While a definite conclusion cannot be drawn from this small number and short period of follow-up, it would seem, as was indicated in the earlier cases, that the sacrocervical ligament alone is capable of producing and maintaining indefinitely adequate support for the cervix and uterus to prevent recurrence of uterine retroversion and prolapse.

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REVISED TECHNIQUE FOR CONSTRUCTION OF THE SINGLE BARRELED COLOSTOMY

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A REVIEW OF THE LITERATURE concerning abdominal colostomy shows that more than 29 per cent of the single barreled colostomies are followed by some type of complication. The complications most frequently encountered in the single barreled permanent colostomy are stricture of the stoma, hernia, prolapse, and perforation of the bowel. The complications following the temporary colostomy, as carried out by the Rankin obstructive resection or the Mikulicz procedure, are not included in the present discussion.

The frequency of complications in the permanent colostomies is thought to be due to the fact that more abdominoperineal resections for carcinoma of the rectum with establishment of the permanent colostomies are performed now than formerly. Birnbaum and Ferrier state that the number of colostomies performed during the past three decades is fourfold greater while the percentage of complications has risen by 15 per cent for the same period. Other factors favoring the increase of complications in the permanent colostomies are as follows: the mortality following abdominoperineal resection has been greatly reduced, and thus the survival time of these patients is longer, allowing complications to develop. The range of time for development of complications varies from a few months to several years following the establishment of the colostomy.

Formerly, we employed the conventional operational procedure for the single barreled colostomy, and we still consider that the principles outlined in this operation are based upon good surgical judgment, except for the establishment of the colostomy stoma. In this procedure the bowel is allowed to emerge several centimeters beyond the skin, and a clamp is applied to the bowel end for support and to prevent the spill of bowel contents upon the wound. The length of time the clamp is applied to the bowel varies from 3 to 4 days according to the degree of pressure of gas in the bowel and the circulation of the bowel. Further support is afforded by placing plain or vaseline gauze dressings between the bowel and the skin. The colostomy is inspected at 12 to 24 hour periods. If the bowel shows increased distention from gas or disturbed circulation, the clamp is readjusted partially to occlude bowel lumen, and a catheter is placed in the open position for drainage. Following removal of the clamp, if inspection shows the protrusion of bowel to exceed 2 centimeters, the bowel is trimmed with the cautery to this level. The fixation of the bowel mucosa to the skin is a delayed union. In many cases, low grade infection develops encouraging formation of scar or cicatricial tissue about the mucous barrel of the bowel, thus rendering the stoma inadequate for normal function.

In a follow-up of the cases in which the conventional measures for the construction of the stoma of the bowel were used, both personal and referral cases, the percentage of stricture of the stoma was found to run high. In approximately one-third of the personal cases varying degrees of stricture of the stoma developed. This was found to be the result of formation of scar or cicatricial tissue about the bowel mucosa and a recession of the supporting wall of the bowel beneath the skin level. In many of the cases the opening into the bowel stoma was so small that it would not admit the passage of the little finger without great difficulty and much pain. We concluded that the stricture of the stoma was the result of the failure of fixation of the supporting portion of the bowel wall (serous and muscular coats) to the skin for a sufficient time to allow firm adhesions to form between the bowel and skin to prevent bowel retraction, thereby leaving the mucous barrel of the bowel to be encroached upon or constricted by the scar tissue from the repair of the wound.

To meet this situation for correction of the strictured colostomy a plastic operation was necessary. This procedure was carried out by excising the scar tissue from the mucous barrel in

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an elliptic fashion, and by fixation of the supporting wall of the bowel (serous and muscular coats) to the supporting layer of the skin (dermis) and approximating the mucous barrel of the bowel to the epidermis. In the cases in which the plastic operation was carried out our results were satisfactory.

The results obtained in the management of instances of strictured colostomy gave us the idea which influenced us to adopt the same procedure for primary colostomy with some modification.

REVISED TECHNIQUE

Spinal anesthesia is the anesthetic of choice, unless contraindicated, when inhalation anesthesia is employed. The anesthetic should be one which gives the maximum degree of relaxation to the abdominal and pelvic tissues.

The incision for the establishment of the colostomy should be independent of the incision for exploration of the abdomen and surgery to the colon and rectum preparatory for the construction of the colostomy. The two incisions most favored are the lower midline or the lower left rectus incision. The advantages offered by the lower midline incision are: (1) it affords a better evaluation of the abdominal viscera for the possibility of involved aortic lymph nodes and liver metastases if the case is one of a malignant lesion of the lower colon or rectum. (2) It aids the mobilization and resection of the lower colon, gives the best exposure of the deep pelvis for rectal resection, affords an opportunity of visualizing the ureters with minimal chance of damage to the ureters and other pelvic organs, and finally the lower midline incision brings into view the open pelvis following the rectal resection for immobilizing the pelvic peritoneum for successful closure of the pelvic floor. The left rectus incision is often used but does not offer the same advantages for carrying out pelvic surgery as the low midline incision.

The main disadvantage or hazard occurs in some cases in which the incision extends upward into the rectus muscle. The stoma then of necessity must be made in the upper angle of the left rectus incision or in close proximity to it.

If the colostomy is established in the upper midline or left rectus incision and infection occurs in the wound, healing of the wound will be interrupted and chances of complications arise, namely, dehiscence of the wound may occur and breaking down of the colostomy resulting in peritonitis from spill of bowel content into the wound, and an unsatisfactory colostomy may result.

In the revised technique the colostomy is established through a separate left rectus incision, splitting the fibers along the medial border of the rectus muscle. The incision for the colostomy is located on a line between the anterior superior spine of the ileum and the umbilicus, after 1 centimeter of skin has been excised in a conical fashion from the midportion of proposed skin incision (Fig. 1).

The colostomy limb consisting of either upper or midsigmoid segment should be of a length to avoid redundancy of the bowel which encourages stagnation of feces in the bowel prior to its emerging through the abdominal wall. On the other hand, it should be of sufficient length to prevent tension to the bowel as it is brought out from the abdomen onto the skin with preservation of a good blood supply to the limb.

The opening in the abdominal wall should be of such size to accommodate the bowel and allow the index finger to pass along the side of the bowel without constriction to the bowel. In the closure of the upper and lower angles of the colostomy wound, we employ interrupted No. 00 chromic surgical sutures for the peritoneum and aponeurosis, No. 00 plain interrupted surgical sutures for the muscle and Scarpa's fascia, and interrupted No. 00 silk for skin.

To aid the support of the bowel as it emerges from the abdomen, multiple epiploic tags of the bowel are sutured to the parietal peritoneum (Fig. 2). In addition to the support offered the bowel by utilizing the fat tags, bringing this tissue in proximity to the opening in the peritoneum will lessen the chance of the bowel becoming adherent to the peritoneum and of encouraging bowel obstruction.

Further support to the bowel is made at the skin level by suturing the supporting portion of the bowel wall (serous and muscular coats) to the dermis of the skin with No. 000 chromic interrupted surgical sutures spaced ½ centimeter apart (Fig. 3). Entering the mucous barrel of the bowel with the suture should be avoided in this procedure. The mucous barrel of the bowel is then sutured to the epidermis of the skin by employing No. 0000 silk as interrupted sutures spaced the same as for the serous-muscular coats (Fig. 4).

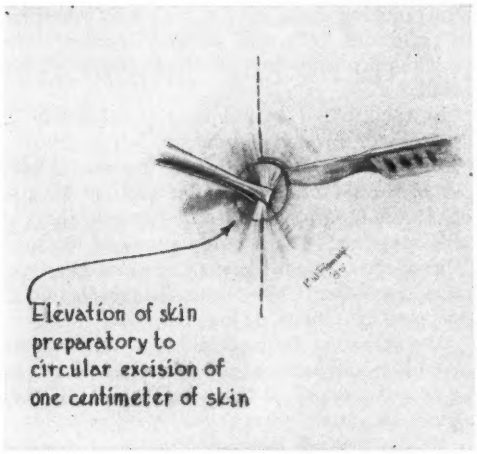

FIG. 1. Proposed left rectus incision below the umbilicus with measures for removal of a conical shaped piece of skin.

No attempt is made to close the hiatus (opening between bowel and lateral peritoneal wall) when the left rectus or the lower midline colostomy stoma is constructed. The lower midline abdominal incision is closed by the usual standard surgical procedure.

The revised procedure for stoma construction gives: (1) better fixation of the bowel wall (serous and muscular coats) to the supporting portion of the skin (dermis) to overcome the retraction of the bowel; (2) preservation of the marginal blood supply to end of bowel; and (3) fixation of the mucous barrel of the bowel to the skin (epidermis) resulting in a primary union of the mucosa to the skin and normal eversion of the mucous barrel.

For a colostomy to function it should admit the index finger with ease and be sufficient in size to pass a normal stool. If it is smaller, partial bowel obstruction will follow, irrigation is rendered difficult, and perforation of the bowel is likely to occur.

The chief objection that might be offered against the revised operation, and this was seriously considered, was the possibility of infection of the wound that might result from the escape of bowel contents, feces, and gas upon the wound before union of the bowel mucosa and skin took place. Such infection did not occur in any of our cases. Doubtless, the chief reason infection did not develop is that in surgical procedures involving the abdomen, more particularly the intestinal tract, there is inhibition or cessation of the peristalsis of the bowel for 2 or 3 days. This condition reduces escape of bowel contents (feces and gas), allowing sufficient time for union to take place between the mucous barrel of the bowel and the epidermis of the skin.

DISCUSSION

For carcinoma of the rectum below the peritoneal line, the abdominoperineal resection offers the lowest incidence of that most distressing of all complications of carcinoma of the rectum —pelvic recurrence.

Following the removal of the rectum it is necessary to divert the colon drainage to the outside by connecting the colon stump to the skin of the perineum or the abdomen.

In spite of the fact that perineal colostomies have been advocated, they do not offer the same advantages as the abdominal colostomy. Lahey commenting upon the perineal colostomy said that, "Since the perineum is in a dependent position to the body, passing a catheter into the bowel meets with difficulty, and pinching the skin about the colostomy to fill the colon is not as satisfactory as the abdominal colostomy." A

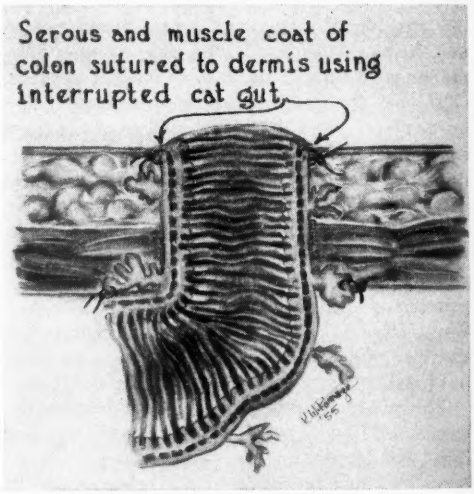

FIG. 2. The limb of the left colon for the colostomy as it leaves the abdomen to emerge through the abdominal wall with fixation of the epiploic tags to the parietal peritoneum and the support given to the serous and muscular coat of the bowel by employing interrupted No. 000 chromic surgical sutures to the dermis of the skin spaced approximately ½ centimeter apart.

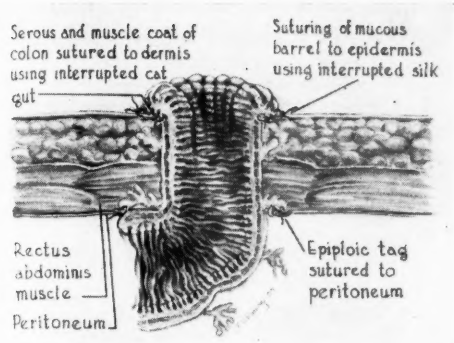
Serous and muscle coat of colon sutured to dermis using interrupted cat gut
Suturing of mucous barrel to epidermis using interrupted silk
Rectus abdominis muscle
Peritoneum
Epiploic tag sutured to peritoneum

FIG. 3. The completed single barreled permanent colostomy after suture of the mucous barrel of the bowel to the epidermis of the skin with No. 0000 interrupted silk sutures.

colostomy placed in the perineum or on the abdomen does not possess any sphincteric control. The bowel movement is best controlled by the size of the opening of the colostomy, one that will admit the passage of the index finger with ease, diet to favor constipation, and irrigation of the bowel to obtain bowel movement.

The site of the abdominal colostomy should be in the most convex portion of the abdomen to permit its best management. The most common site for the abdominal colostomy is either in the midline of the abdomen beneath the umbilicus or to the left and below the umbilicus, opposite the rectus muscle. Either site will make a satisfactory location for management by the patient, and too, the convex or the most prominent part of the abdomen offers the best location for wearing an elastic band 6 to 8 inches in width for support of the colostomy and maintaining the position of a pad of gauze or tissue paper to care for the mucous drainage from the bowel stoma. Wakeley employs the left paramedial incision, going through the anterior and posterior rectus fascia, withdrawing the rectus muscle laterally, extending the skin incision upward and medially to the umbilicus, and removing the umbilicus. His reason for removal of the umbilicus is to prevent infection of the umbilicus with subsequent sepsis caused by the drainage of the bowel content upon the umbilicus. This excision of umbilicus is recommended by many of the men who employ the midline incision. The late T. E. Jones, and Schaerrer and Campbell, preferred to bring the bowel out through the lower midline incision beneath the umbilicus. The advantage claimed by placing the colostomy in the midline of the abdomen is that the bulge of the colostomy can be more easily camouflaged.

Formerly, the left inguinal colostomy was looked upon with favor by a good percentage of the surgical profession, but the enthusiasm for the inguinal colostomy was soon overcome for the following reasons: due to its low position in the groin, it was found more difficult for the patient to irrigate the colostomy; formation of secretion in the groin region made it less sanitary; to bring out the colostomy in the inguinal region weakened the inguinal tissues with chances for subsequent development of inguinal hernia; and, finally, the low position of the bowel in the abdomen formed a shelf inside the abdomen encouraging a loop of bowel to become obstructed there.

The most popular site for the abdominal colostomy has been to the left and below the umbilicus; the muscle is retracted laterally or is split as an independent incision in addition to the midline incision which is employed for exploration of the abdomen and removal of the rectum. In case a lower left rectus incision has been made for exploration and removal of the rectum, the colostomy is brought out through the upper portion of the left rectus incision. In addition to the location on the abdomen, a satisfactory abdominal colostomy depends on other factors. The bowel should be of sufficient length to leave the abdominal cavity in a straight line, free of tension which interferes with the blood supply to the bowel. The straighter the loop of the colon, the easier it will be for the patient to

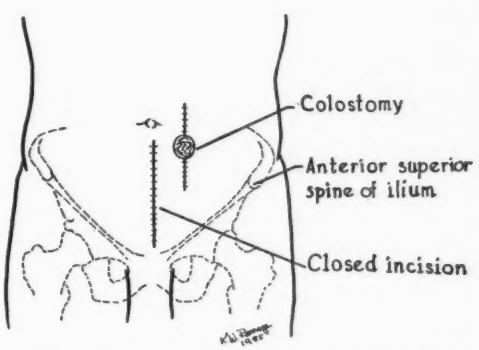

FIG. 4. Schematic diagram showing location of colostomy in its relation to umbilicus and anterior superior spine opposite left rectus abdominis muscle.

insert the catheter for irrigation of the colostomy. A long redundant loop of the bowel is to be avoided as this leads to colon stasis and more difficulty is encountered in emptying the bowel by irrigation. Another point of most importance in all colostomies is to be sure that there is adequate blood supply to the section of the colon which is to be implanted in the abdominal opening as well as preservation of the marginal blood supply to the end of the bowel.

The management of the lumbar gutter will depend on the location of the colostomy. When the colostomy is placed in proximity to the left rectus muscle or the midline of the abdomen, the hiatus between the bowel and the abdominal wall is too large for closure. Breidenbach and Slattery state that they have not encountered obstruction due to the small bowel entering the opening between the colon limb and the abdominal wall, so called lumbar gutter, and do not consider closure of the space an important point. Miles, who was the first to recommend the abdominoperineal resection for carcinoma of the rectum, and other pioneer surgeons in the field of colostomy construction have advised against closure of the lumbar gutter. We believe that only in cases in which the bowel is brought out through a stab wound in the left lower quadrant or upper inguinal region should consideration be given for the closure of the gutter. The reason for closure of the gutter in this type of case is that bringing out the bowel for the colostomy in this location will create a narrow or small gutter that may encourage a loop of small bowel to become trapped in the opening with resulting obstruction of the bowel. The closure of such a gutter should be complete. Colcock states that an incomplete closure is worse than leaving the gutter wide open. In the approximation of the bowel to the peritoneum for the closure of the gutter, tension or traction to the bowel limb is to be avoided. This is best accomplished by incising the peritoneum distal to the white line of the descending colon and sigmoid segment sufficiently cephalad to free the bowel completely from its peritoneal attachment. If necessary to obtain relaxation of the bowel, the incision should extend up to, and include the fixation of, the splenic flexure of the bowel. Closure of the space should be carried out by suturing the edge of white line of the bowel to the lateral peritoneum.

SUMMARY

1. The high incidence of complications following the conventional operation for construction of the single barreled colostomy stoma is conclusive that this procedure fails to meet the requirements for a satisfactorily functioning colostomy.

2. Stricture of the colostomy stoma is responsible for the majority of complications occurring in the single barreled colostomy.

3. The revised operation for the construction of the bowel stoma herein described prevents stricture.

4. Perineal colostomies are to be condemned.

5. The colostomies established by a left rectus incision, either splitting or retracting the muscle laterally, or by a midline incision below the umbilicus are the most favorable for management by the patient.

6. Requisites for a satisfactory colostomy are a straight bowel limb as it emerges from the abdomen through the abdominal wall, the bowel limb to be free of tension and constriction as it passes through the abdominal wall, and maintenance of good blood supply to the bowel.

7. Supporting the bowel wall to the dermis of skin to prevent recession of bowel and eversion of the mucous barrel to epidermis of skin, as outlined in the revised operation for stoma construction, gives the best results.

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The Book Shelf

A MEDICAL TRUANT

ESTHER H. VINCENT, Evanston, Illinois

IN THE PLEASANT PROTESTANT CEMETERY in Rome, near the Pyramid of Caius Cestius, lie the graves of Keats and Shelley. When the Pyramid was built no Roman of that day could possibly have foreseen that centuries hence it would serve as a landmark for the graves of a couple of young poets from the remote and barbarous isle of Britain. Nor in 1821 could the dying Keats have had the slightest idea that his self-chosen epitaph, "Here lies one whose name was writ in water," would some day acquire solid fame.

Tom Keats was a hearty West Country lad, who came up to London, got a job at a livery stable, married the boss's daughter, and shortly succeeded to the profitable business. Brown-haired, hazel-eyed Tom was a small, lively man, blessed with sense and intelligence, and with an air of good-natured respectability. Frances Jennings Keats was tall and well formed, impulsive and fun-loving, and passionately devoted to her children, especially to John, the eldest, although she imprudently delivered him 2 months early (October, 1795). There were 4 other children, George, Tom (always delicate), Fanny, and Edward, but little Edward did not survive infancy. At first they lived at the stable, at the sign of the Swan and Hoop (now 85 Moorgate St.), but they later moved to a house in Craven Street, City Road, the facade of which is now shown at the South Kensington Museum as an example of Early Georgian classic architecture.

The boys were sent to John Clarke's school at Enfield, 10 miles north of London (John Gilpin's route), a red Georgian mansion, where John made friends with the master's son, Charles Cowden Clarke, the future Shakespearian scholar. When John was 9 years old, his father was killed by a fall from a horse (skull fracture), and his mother's too speedy second marriage ended in as speedy a separation. But the family was well cared for by the Jennings, who had retired to a pleasant country life at Edmonton.

Schoolboy John showed great promise, and his terrier-like courage made him a popular fighter. He was a beautiful child, with red-gold hair, vivacious, and pugnacious, but at the same time generous and affectionate, and usually happy, but subject to occasional fits of melancholy. When he was 14, his mother, long a victim of chronic rheumatism, died of consumption. John spent hours caring for her and was exposed to the disease. It is quite possible that all three boys were infected at this time, and delicate Tom was not able to defeat the bacilli. George and John, with their robust constitutions, were well able to furnish the necessary resistance. It may well be that John would have continued in health had it not been for later physical exhaustion and further exposure.

Grandfather Jennings died and so did his son, a distinguished naval officer, and Grandmother Jennings was getting old. So she put her money in trust for the children, and the children under the guardianship of a merchant and a tea dealer. The merchant soon faded out of the picture, but tea dealer Richard Abbey did his best to look after the children, taking little Fanny into his own home, keeping George and Tom at school,

and apprenticing 15 year old John to a country doctor, Thomas Hammond of Edmonton. Hammond's house, said to be still standing, has always been occupied by a physician. Here John held the horse, packed the saddlebags, compounded pills, drew teeth, vaccinated, and bled patients. His grandmother lived on the same street, and he could easily walk the 2 miles to see Cowden Clarke at Enfield. But at the end of 4 years, Hammond dismissed him as a lazy, idle fellow, always reading and scribbling. Despite this, his good-intentioned but unperceptive guardian insisted that John should continue his medical studies.

In 1815 Keats entered Guy's Hospital as a student. At this time Guy's and St. Thomas's were still more or less united, freely exchanging equipment and personnel, both depending on "Resurrectionists" for dissection material, neither attempting organized instruction but merely giving the students opportunities to learn what they could. The young men "walked the wards" and took notes if they felt like it. School examinations were unknown, but, in order to take the licentiate examinations at Apothecaries' Hall, candidates had to present certificates of attendance at lectures on anatomy, medicine, materia medica, and chemistry.

The dressers accompanied the surgeons on their rounds, carrying their "wand of office," a tin box containing bandages, and were aided in changing dressings by the untrained precursors of nurses. Dressings had to be changed frequently, owing to prevailing septic conditions. Each dresser had outpatient duties, also, until early afternoon, and each man witnessed as many surgical operations as possible, enduring the unanesthetized groans and shrieks, crowding close and crying "Heads, heads," when some other pusher got in his line of vision. Dressers took in rotation a week's emergency duty, living in the hospital and paying their own board.

In the office of Guy's Medical School may still be seen the new student entry book in which Keats signed his name, but the name itself has been cut out by an autograph thief. However, his name appears in two other books as a pupil and dresser. It is interesting to note that in the entry book, the name next after Keats is that of the painfully notorious John White Webster, murderer of George Parkman of Harvard Medical School.

Keats was dresser to William Lucas, Jr., one of the three surgeons then at Guy's, the others being Thompson Forster and Astley Cooper. Pleasant, old Forster seldom operated. Good-natured Lucas was an awful bungler, but, fortunately, rarely operated without the brilliant Cooper. Oddly enough, Cooper took notice of little Keats (just over 5 feet) and showed him some attention. Cooper came to Guy's on Tuesdays and Fridays, stayed for 30 or 45 minutes, and went over to St. Thomas's. His evening surgical lectures were jammed with adoring students.

Keats was one of those painstaking pupils who took notes, and his worn, limp, brown leather notebook may still be seen, its price-mark, 2/2, still plain. He wrote his elementary anatomy notes in a large, neat hand on alternate pages, but, curiously, he scribbled his physiology notes without order on whatever page to which the book happened to fall open. He always recorded important, fundamental points, and seems to have been soundly trained in the medical lore of his time. His jottings include phrases about the newly discovered red blood corpuscles, needle-pricking to relieve dropsy, and the effects of digitalis. His humor shows up when he notes that "In disease Medical Men guess, if they cannot ascertain a disease they call it nervous." There are points of resemblance between Keats's physiology notes and Cooper's *Lectures on the Principles and Practice of Surgery*, since Cooper frequently drew examples for his book from his physiology class lectures.

The physicians of Keats's time at Guy's were James Curry, Alexander Marcet (the Swiss chemist), and James Cholmeley. "Calomel" Curry was a fluent and brilliant lecturer, although it is said that if his diagnosis of liver disease was unsubstantiated at autopsy, he claimed a cure by calomel. Cholemely, a capable physician, remembered Keats for his intelligent observations.

Keats passed his licentiate examinations with honors, although he prepared for them in the shortest possible time, 10 months, and took them at the youngest possible age, 21. However, he did not really like medicine, and how the future author of the "Ode to a Grecian Urn," lived through the "sights and sounds and shrieks unholy" of the operating room is a miracle. One day he opened a temporal artery with great skill, but while operating "there came a sun-

beam into the room, and with it a whole troop of creatures floating in the ray, and I was off with them to Oberon and fairyland." Having a deep sense of responsibility, he resolved not to enter a profession for which he believed himself unfitted. Not for him was the passionate interest in the workings of the human body in either a scientific or a philosophic sense. He had worked hard at medicine, but he quit without ever having practiced. Nevertheless, there is a ward at Guy's named for him.

He had written his first fine sonnet, "On first looking into Chapman's Homer," in the summer of 1815. However, long before that he had believed himself permanently inoculated with poetry, the inoculation dating from his first exposure to Spenser's "Faerie Queene." His decision to abandon medicine met with the expected opposition of his guardian, Mr. Abbey, but Keats joined his brothers at their Hampstead lodgings and happily began to write. He had already met Shelley and Leigh Hunt, both of whom took an interest in him, especially Hunt, who published his poems in the literary weekly, *The Examiner*. His first slender volume of poems came out in 1817.

Most of what we know about Keats comes from his voluminous correspondence, and it is in October of that year that he first refers seriously to his health. "The little Mercury I have taken has corrected the poison and improved my Health, though I feel from my employment that I shall never be again secure in Robustness." At this time mercury was often used for indigestion and minor infections, and it is evident that whatever illness Keats then had was temporary, because it was during this winter that he spent an hour thrashing a big bully of a butcher who had been mistreating a kitten. Keats was often away visiting friends at Oxford, Stratford, and other places and apparently led a healthy, active life.

George had now married and emigrated to America, leaving John to care for Tom, who was slowly succumbing to tuberculosis. The two youths lived close together in small quarters with windows shut. In June of 1818 John suffered a further minor indisposition and writes that "Doctor says I mustn't go out." During the summer Tom seemed better, and so John left him in order to accompany his genial merchant friend, Charles Armitage Brown, on a walking tour of the English Lake District and of the

FIG. 1. John Keats. Bronze cast from a life-mask. (From the National Portrait Gallery, London.)

Scottish Highlands, including the islands of Mull, Staffa, and Iona. Tramping the hills and dales in rain and fog, sleeping in damp smoky huts, eating poor food, Keats wore himself out. Climbing Ben Nevis, they "groped and struggled in a toilsome mist," and on arrival at Inverness, Keats had a "confounded toothache" and sore throat. Ragged and exhausted he reached London to find Tom much worse. Dr. Sawry, Tom's doctor, treated John's sore throat by keeping the patient indoors and prescribing mercury. It did not interfere with his activities much, and the soreness soon disappeared without recurrence until after Tom's death in December.

Keats then went to live with Brown in a double house, Wentworth (now the Keats Museum), the other side of which was occupied by their friend, Charles Wentworth Dilke, the antiquary and critic. During Brown's absence on the Scottish tour, Fanny Brawne and her mother had lived in his house, and after his return they moved to a house nearby. Keats fell deeply in love with attractive 17 year old Fanny, and later became engaged to her. Some of Keats's biographers think that the lively, fair-haired maiden was a shallow flirt and that her charm for him was merely a matter of propinquity, but others read into her letters a warmth of personality and an intelligent ap-

preciation of his genius that could have made her a stimulating wifely companion.

Keats was being savagely attacked by the chief literary reviewers of the day, who, for some reason, took a dislike to Hunt's "Aesculapian jackal" and were blind to his worth. As long as he was feeling well, Keats took the stinging criticism stoutly and bravely, having a well founded belief in himself. With declining health, however, he found it hard to bear. He had money troubles, also, which caused him much confusion of thought. Mr. Abbey had not handled his trust too efficiently, and when Tom's share of the family funds came to be divided between John and George, the resulting amount was disappointing. Keats brooded over a possible necessity of returning to medicine. He thought of going to Edinburgh for further study, and then starting practice, or of taking a job as ship's surgeon on an Indiaman or on a ship bound for South America. He also considered becoming a journalist or a playwright. None of these schemes, however, ever got beyond general planning. He finally went to the Isle of Wight for part of a summer and wrote in order to give poetry a fair trial. He had periods of well-being but sometimes a sense of general ill-being. During the fall he made no complaint, but he was afraid of the coming winter's cold.

In February of 1820, on a day of damp, penetrating chill, he left home without an overcoat and rode to London on the outside of a stage-coach. On the way back, coughing, flushed, and fevered, he spit up something in the dark, but could not distinguish what it was. Once in the house, he examined his sputum by the light of a candle, noting fearfully its bloodiness. Dr. Robert Bree diagnosed the case as one of nervous irritability, systemic weakness, and poetic excitement. "'Tis not yet consumption," wrote Keats. Nevertheless, the doctor prescribed prolonged bedrest. After a monotonous period in his room, Keats repaired to the parlor sofa where he could watch the passers-by. There is some mention of heart palpitations at this time, but not afterward. Pampered with tenderness and invalid food, Keats writes of smearing currant jelly on a page of Brown's copy of *Ben Jonson*, and of vainly trying to lick it off. He felt better in the spring, again indulging in his propensity for long walks. Brown, believing the crisis passed, rented the house, and betook himself to Scotland for another walking tour.

Keats took lodgings in Kentish Town, near the Hunts, and worked on the poems that he had written since *Endymion*, the poems making up the slim, little volume of verse on which rests his fame as a great English poet.

In June he began to have repeated violent hemorrhages, and Leigh Hunt, finding him helpless, took him home. There he lay, white as the bedclothes except for the hectic flush on his thin cheeks. The Hunts, kindly, though feckless, took care of him as well as they could, in spite of their riotous houseful of children. George Darling, a general practitioner of Russell Square with a large clientele among writers and artists, was his new physician. In consultation with him was kindhearted, generous William Lambe, a Cambridge doctor and a Fellow of the Royal College of Physicians. He was a bit of a fanatic on the subject of vegetable diets. He also believed that disease originates outside of the body, but attributed it to undistilled water. He had some good theories but did not test them. Dr. Bree, not too successful in Keats's last attack, was not sent for, possibly because of the expense.

Poor Keats was now almost paralyzed with despair. He had none of the *spes phthisica*, that optimism so often characteristic of the tuberculosis victim. He turned misanthropic and suffered acute jealousy in regard to the innocent Fanny. His love for her was always oddly feverish—he could be calm enough in her presence or when she was far away, but her near though unseen presence could rouse him to a frenzy. Through a servant's error, one of her letters was belatedly delivered and with the seal broken. This episode so upset Keats that he left the good Hunts and went to stay with the Brawnes, who now lived in the old Dilke side of Wentworth House. Mrs. Brawne gave him the best of motherly care, but she was very much aware of the tragedy of having her young daughter engaged to a dying man.

A period of tranquillity ensued, and when Keats was so improved as to be able to crawl around, Dr. Lambe prescribed the well worn but usually futile formula of a winter in Italy. With the artist, Joseph Severn, as a companion, Keats started for Naples. As his vessel sailed down the Thames in September of 1820, it passed, unawares, the ship of his friend, Brown, returning from Scotland.

Contrary winds in the English Channel, storms on the Bay of Biscay, stop-and-search by

a Portuguese man-of-war, quarantine at Naples, all added to the length of the voyage. The stifling air of the cabin, the indifferent food, and the presence on board of another consumptive added to Keats's misery. The two young men went up to Rome by carriage, enduring many bad inns and much poor food along the way. Shelley had invited Keats to stay with him at Pisa, but Keats and Severn took rooms in a house on the right of the Spanish Steps, engaged for them by James Clarke. Dr. Clarke had taken his medical degree at Edinburgh, was a popular physician with British royalty, and was later chosen to write the article on "Tubercular Phthisis" in the 1834 *Cyclopedia of Practical Medicine*. He did not even suspect the existence of the tubercle bacillus, but he did suspect that the coughing up of blood indicated the destruction of a blood vessel. He thought this rare, however. He also considered that the disease must have progressed quite a bit preceding the appearance of the symptom, and furthermore, he believed in the efficacy of fresh air. Nevertheless, he did not appreciate the gravity of Keats's case, thought it only a slight affection of the lungs and prescribed short, easy horseback rides. Keats walked about some on the Pincian Hill and took up the study of Italian.

On December 10 he had new hemorrhages, losing blood by cupfuls, and he burned with fever. The devoted, kindly Severn was nurse, cook, and servant day and night, except toward the last when an English nurse came in for 2 hours every other day. It was Severn who took from Keats the small bottle of laudanum he had brought from London. Dr. Clarke came several times a day, and placed Keats on a starvation diet of 1 anchovy, a bit of bread, or a pint of milk. A slight rally in January, 1821, gave him opportunity once again to walk on the Pincio, but with recurrent hemorrhages he took to his bed. His chief comfort was listening to Haydn Sonatas and to the reading of Jeremy Taylor's *Holy Living and Dying*. He felt bitterly the strain of his burden on his friend. Otherwise, he had lost all interest in life, living, as he said, "post-humously," and feeling the flowers already growing over him. Death came in the early morning of February 23.

Dr. Clarke and an Italian physician performed the autopsy. The lungs were entirely gone. No mention was made of any laryngeal pathology, thus disposing of the idea that Keats's earlier recurrent sore throat indicated tuberculosis of the larynx. Such a disease would have progressed rapidly with hoarseness, voice loss, and pain in swallowing. Keats's sore throat troubled him only intermittently and finally not at all.

Like all patients of the time, Keats was bled severely, even after his hemorrhages. In those days doctors sometimes bled people to death. There is at least one case in which a doctor was sued for malpractice for not bleeding a man who had pneumonia. It was not until 1830 that Marshall Hall showed the folly of indiscriminate bleeding in his book, *Research Principally Relative to the Morbid and Curative Effects of Loss of Blood*. Laennec's *De l'Auscultation Médiate*, 1819, was unknown in England until after 1821, and, of course, there was no knowledge of thermometers, microscopes for sputum, or x-rays. Koch did not discover the tubercle bacillus until 1882. The physicians of Keats's day were not any more stupid than they are now, but medical research was slow.

In tramping the Lake District with Brown, just prior to their Scottish tour, Keats met and talked with Coleridge. In 1832 Coleridge, then a sick, old man, said that he had felt death in Keats's hand that day when he shook hands with him, but that he had been in his company for only a minute or two. Keats, at the time, however, reported no handshaking at all, but did say that he had walked 2 miles with Coleridge and a friend at a slow pace. The memory of an old opium-taker is not likely to be accurate. Keats had no symptoms of tuberculosis until after Tom's death.

Medical allusions in his writings are infrequent. In a letter to his sister, Fanny, he describes "one Richard Bradshaw, a notorious tippler" with this prescriptive simile of balance, ℥. In the "Eve of St. Agnes":

> "Angela the old
> Died palsy-twitch'd with meagre face deform;"

In *The Cap and Bells* Magician Hum is "glutted like a leech." In *Endymion* the phrase, "And warm with dew at ooze from living blood," probably refers to the serum separating from the clot when blood is allowed to stand after venesection. In *Otho the Great* he writes of "melancholy blood" in the sense of blood infused with black bile, a survival of the old idea of humoral pathology, and in the lines following he refers to an old man's "heavy pulse" as

"painful, clog up and stagnate," a condition of arterial obliteration and thrombosis. A more apt simile of lice hiding in the wrinkles and creases of old, dirty skin cannot be found than in the phrase, "crannied vermin."

Keats was of striking appearance in spite of his small stature. His head was well shaped, his hair thick and curling. His rather long, delicate face was arresting, the upper lip protruding just a little. His chin was bold, his large, hazel eyes, darkly glowing. The life-mask in London's National Portrait Gallery is hauntingly attractive. By nature he was essentially upright, courageous, and affectionate.

Near the close of his life he wrote, "If I should die, I have left no immortal work behind me—nothing to make my friends proud of my memory—but I have lov'd the principle of Beauty in all things and if I had had time I would have made myself remembered." He did not have much time, it is true, but few are so well remembered. To paraphrase a line from his "Grecian Urn":

Forever shall we love and he be fair.

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Reviews of New Books

IN THE PREFACE to the first edition of the *Manual of Human Dissection*, Shearer expressed the hope that his laboratory guide would prove useful to those who, like himself, had come "to feel that, with the serious decrease in the number of hours allotted to the subject of gross anatomy in medical schools of recent years, the admirable but lengthy guides at present available are not entirely satisfactory."

The success of the original work, equalled by that enjoyed by each of the successive editions, attests to the wisdom of Shearer's decision and of Charles E. Tobin's good judgment in improving the original text upon the basis of his own experience and that of his friends.

Tobin wisely and modestly informs both teacher and student that the dissector should use the book[1] for the purpose for which it was written, that is, as a manual of dissection—the information thereby obtained to be "supplemented by collateral reading in one of the standard descriptive texts if the student is to obtain a thorough understanding of human structure."

The volume contains 79 figures, engraved from graphically simple line drawings. Although offering essential and convenient aid to the dissector, their employment should be supplemented by regular recourse to more ambitious series, of atlas proportions.

The general arrangement of the contents is as follows: chest, axilla, neck, back, thorax, abdomen, perineum, superior extremity, and inferior extremity. This succession does not, however, bind the user to sequence presented; the text is written in such a way that the instructor may handily alter the order to fit the curricular needs of his department.

Everywhere the instructions are succinct and authoritative; the placement of figures is convenient; the printing of important terms in bold-face letters is a typographic device serviceable either for first reading or for any subsequent review.

The *Manual of Human Dissection* has enjoyed the commendation of many reviewers. To predecessors in recognition of its merits we enthusiastically add our own. —*Barry Anson.*

IT WOULD SEEM IMPOSSIBLE to sustain a reader's interest in fluoroscopy for the span of 741 pages. Yet, the author of the book *Fluoroscopy in Diagnostic Roentgenology*[2] has, to a great extent, achieved this task. The book is well printed, well written, and well illustrated. The approach to the subject is honest and, on the whole, not embellished by such vague and abstruse and often unconfirmable fluoroscopic findings as so often appear in the fluoroscopic literature. It is obviously the product of a large practical experience in fluoroscopy.

The chapter on the "Development of Fluoroscopy" is interesting in the historical perspective that it gives. Special tribute is paid to Guido Holzknecht and the Viennese X-ray Institute that he founded. The contributions of Cannon, Williams, Manges, Carmen, Kirklin, and Chamberlain in this country are especially mentioned. Interesting tidbits are scattered throughout the book. In this chapter, for instance, we find out the origin of the term "flat plate."

The chapter on "The Conventional Fluoroscopic Image" contains a useful and practical discussion of dark adaptation and accommodation, and an interesting graph shows the relationship of screen brightness and milliamperage.

A short discussion is given on "Electronically Amplified Fluoroscopy," and this subject is briefly touched on in various parts of the book. A short but useful chapter is devoted to "The Hazards of Fluoroscopy," and numerous methods of localizing opaque foreign bodies are described in the chapter on "Foreign Bodies and Their Localization." It would be interesting to know how successful the "monocular cryptoscope" was in localizing foreign bodies.

The clinical chapter on myelography is scanty, and the reviewer can see little benefit in the use of fluoroscopy during encephalography or ventriculography. A long chapter is devoted to "Fluoroscopy of the Chest," but to this reviewer much of this material seems to be rather forced and the method in many instances is inferior to radiography. Numerous of the excellent illustrations are examples of conditions that are much more accurately evaluated by radiography than by conventional fluoroscopy. The illustrations and text are clear; and it is fascinating to read, for instance, in the account of bronchial obstruction, about the phenomena of Holzknecht-Jacobson, or Holzknecht-Hofbauer, of Hitzenberger, and of Manges.

In the section dealing with cardiac fluoroscopy, some statements are made that create an inaccurate impression. For instance, when a hypoplastic appearance of the thoracic aortic shadow is discussed, examples quoted include patent ductus arteriosus (practically never associated with a hypoplastic aorta) and the poststenotic portion of the coarcted aorta (which is practically always dilated). The statement that in infants with atrial septal defect there is a right-to-left shunt, whereas this becomes a left-to-right shunt in adults, is inaccurate. Another statement, long disproved, namely that the so-called hilar dance is the result of pulmonary regurgitation, is included in the discussion of the Eisenmenger syndrome.

In the reviewer's opinion, the main value of the clinical section of this book lies in the excellent presentation of the fluoroscopic method in gastrointestinal examinations. The text is clear and practical, the illustrations good, and irrelevant material is not included to burden the reader. More extensive discussion of such pediatric entities as vascular rings and

[1] SHEARER'S MANUAL OF HUMAN DISSECTION. Edited by Charles E. Tobin, Ph.D. 3rd ed. 287 pages, 79 illustrations. $6.00. New York, Toronto, London: McGraw-Hill Book Co., Inc., The Blakiston Division, 1955.

[2] FLUOROSCOPY IN DIAGNOSTIC ROENTGENOLOGY. By Otto Deutschberger. With an introduction by Frank J. Barrelli, M.D., F.A.C.R. 771 pages, with 888 illustrations on 523 figures. $22.00. Philadelphia and London: W. B. Saunders Co., 1955.

tracheoesophageal fistulas could have been given, and it is surprising that these conditions are not illustrated. It is amusing to find that the configuration of the stomach in certain cases of cascade stomach has inspired the designation "obscene stomach" (Figure 397) because the appearance resembles the scrotum and phallus in profile. It is doubtful that this term will ever attain more than intradepartmental use.

—André J. Bruwer.

CLINICIANS AND STUDENTS interested in the diagnosis and treatment of vulvar disorders will find the fourth edition of Elizabeth Hunt's *Diseases Affecting the Vulva*,[3] like the previous editions, a concise and authoritative textbook. The book contains relatively few pages and is written on a limited subject, yet it has a valuable place among medical texts. Written by a dermatologist, it understandably places emphasis on diseases of the vulvar and perineal skin. More space is devoted to lichenification, intertrigo, psoriasis, seborrheic dermatitis, and lichen planus of the vulva than is given to the various types of vulvar carcinoma. As many pages are allotted to nonvenereal ulcers of the vulva as there are to venereal diseases. Hunt has very wisely concentrated attention on those affections of the vulva which are usually omitted in gynecologic texts and which are discussed only briefly in textbooks of dermatology. As the reader explores the author's material he is reminded that the integument of the vulva is, after all, a part of that covering the rest of the body. The affections which are peculiar to it, including malignancies, are few in number as compared with those which are neither specifically genital nor cancerous.

A number of chapters in the book describe the vulvar lesions which develop as part of various generalized dermatologic conditions and show how certain characteristics of the vulva and its environment tend to modify the appearance of the lesions in it. An important section appropriately points out that the vulva also frequently exhibits morbid changes which are secondary to systemic or constitutional disorders. The chapters on affections of the vulva in childhood merits special commendation. The material on herpes gestationis, new in this edition, is a valuable addition.

For this reviewer the author's discussion on the interrelationship of leucoplakic vulvitis, (which is also called lichen sclerosus vel atrophicus) kraurosis, and leucoplakia of the vulva tends to further becloud a subject which could have added light thrown on it. The use of topical adrenocorticosteroid substances in neurodermatitis and other affections of the vulva is not discussed. Clinicians in the United States who are not dermatologists may have some difficulty with the prescriptions for local treatment which are written in abbreviated Latin.

The 17 full color plates of vulvar lesions are unusually fine as regards both photography and printing. They are supplemented by a number of black and white illustrations depicting the gross and histopathologic appearance of many of the diseases discussed in the text. *—John W. Huffman.*

[3]DISEASES AFFECTING THE VULVA. By Elizabeth Hunt, B.A., M.D., Ch.B. (Liverp.). 4th rev. ed. 236 pages, with 47 illustrations and 17 plates in color. $9.00. St. Louis: C. V. Mosby Co., 1955.

THE SEVENTH EDITION of *Christopher's Minor Surgery*[4] is not a dictionary of surgical entities. It is not a book of recipes for the inexperienced to try when supervision is lacking; nor is it a part of a system of surgery or a reference book with annoying interruptions giving sources of material from endless lists of authors. It was not written to include every surgical condition that might conceivably be handled in an office or a dispensary; nor does it exclude the problems which can be treated to greater advantage within the hospital.

The cover and title page do not indicate it, but this is a textbook. It is one which will have its greatest usefulness among medical students who are just beginning their clinical training in surgery and are working in the outpatient dispensary. Since the book is in its seventh edition, its title is likely to go on unchanged, but the authors are aware of and have done something about a very important fact which influences a student's interest in his work.

The so-called major surgical diseases and their technically detailed surgical management may be highly entertaining for the student beginning his clinical training, usually in the junior year. With the repetition of these favorite stock subjects until the end of the senior curriculum the interest is lost and much useful information is given without appreciation. Time is wasted in the hours of repetition, and hundreds of entities not infrequently seen in surgical practice go without emphasis because of the crowded curriculum.

This surgical text for a surgical dispensary does not compete with texts used in the more advanced courses. The authors have omitted everything possible which would seem to belong to the category of major surgery in both diagnosis and therapy. The subjects chosen generally include such conditions which are visible, palpable, and with good objective or subjective findings to make diagnoses possible on the ambulatory patient. Selection is also affected by therapeutic measures which are generally brief, rather simple to perform, and with little danger to the patient.

The arrangement of the book is in eight parts and twenty-two chapters. The organization of the material may be clarified by considering, by way of example, Part 1, called "General Considerations." Five chapters are devoted to the subject. One chapter discusses the equipment generally needed for minor operative work. Another chapter treats of surgical considerations, actually emphasizing fundamentals in technique. A third chapter is on "Bandaging and Dressings" followed by "Anesthesia and Resuscitation." The fifth chapter deals with "The Surgical Resident," and one will agree that the moment is well chosen for telling students how graduate training is conducted to accomplish what it does.

An effort is made to avoid encroachment upon major problems. For example, Part 4 is titled "Alimentary Tract." The two chapters devoted to the discussion take up the diseases of the mouth and the anus, perianal, and rectal regions. Both are within

[4]CHRISTOPHER'S MINOR SURGERY. Edited by Alton Ochsner, M.D., F.A.C.S., and Michael E. DeBakey, M.D., F.A.C.S. 7th ed. 547 pages, 251 illustrations. $9.00. Philadelphia, London: W. B. Saunders Co., 1955.

reach of the eye and the examining finger. The intervening part of the alimentary tract is left to other books.

Although one would like to comment on each chapter, a review of this type of book will hardly permit it. It should be said that each chapter is written by a different author who is now or formerly was a member of the Department of Surgery of the Tulane University School of Medicine. It is possibly this arrangement which is responsible for the very limited overlap or repetition of related subjects in the different chapters.

The text is beautifully written. The material is presented in a way that it is never so verbose that it is tiring, nor so concentrated that the reading becomes laborious. The approach is strictly introductory for the student entering a new field for the first time, and it is splendid orientation. There are numerous line drawings and photographs strikingly reproduced to illustrate points in the discussion. With approximately 500 carefully selected pages in the book it is predicted that the students who buy it will read it, and that as they advance in their surgical training, they will refer to it many times. —*Jean M. Stevenson.*

IT IS ALWAYS A PLEASURE to read a good book, and an easy task to write a review of it. *Pain*[5] is a good book. Furthermore, it is the first book of such extensive and definitive nature concerned with the study and clinical treatment of pain to have appeared in more than a generation.

The book is divided into three parts. "Fundamental Aspects" deals with the physiologic and psychologic aspects of intractable pain, and the chapters on the psychologic factors, ably written by Stanley Cobb and Frances J. Bonner, greatly enhance one's understanding of the patient and his problem. "Surgical Technique" covers diagnostic and therapeutic nerve block, peripheral neurectomy, rhizotomy, tractotomy, cerebral operations, and sympathectomy. "Treatment of Specific Painful Conditions" is a mine of information, ranging from discussions of the treatment of phantom limb, causalgia, and trigeminal neuralgia, through cephalic pain, to the pain of the various viscera. Gratifying results, complications, disappointments, and personal errors are frankly, honestly, and intellectually discussed in just the manner which the neurosurgical world has come to expect of these well known authors. The current literature on the subject of pain has been combed, sifted, interpreted, evaluated, and effectively used by the authors, in their search to better understand their own painstaking efforts. The chapters on the physiologic basis for pain might well be required reading for all students of neurology and neurosurgery.

In keeping with Thomas' tradition, the book is beautifully printed and illustrated, fitting the rich content. It is a book based on personal experience and obvious clinical and experimental study. To quote Geoffrey Jefferson in the foreword, "this is no 'scissors and paste' affair." More than that, it is a hopeful and inspiring book on a subject all too frequently considered hopeless by a large number of internists, gynecologists, and general surgeons.

—*John Martin.*

THE TEXT OF THE VOLUME entitled *Pathology*[6] by Peter Herbut is presented in compact, terse sentences to outline the basic concepts of pathology for all students of medicine. Much of the material is condensed from the companion volumes of Surgical, Urologic, and Gynecological and Obstetrical Pathology already written by Herbut.

As indicated in the preface, a rigid pattern is followed. Controversial material is eliminated while well established concepts are presented.

The introductory chapter defines pathology and elaborates on its more recent meaning. A brief discussion on the autopsy follows. There could, perhaps, have been some comment on what is now understood as surgical pathology. Weights and measurements of organs are listed.

Space is allotted to the general principles of the field but as soon as possible the author passes on to special pathology. When so doing, the pattern is congenital anomalies, degenerations, inflammations, physical disturbances, and tumors. With this, the reviewer is in hearty accord.

Where possible there are rather clear cut paragraphs on obscure entities delineating the concepts.

Photographs of gross and microscopic specimens are well selected. There are reproductions of roentgenograms and also diagrams. Clinical material is used along with laboratory data to outline certain subjects. All organs and tissues are included in the various chapters.

The print is clear and is not broken into two columns. Paper and binding are most satisfactory.

This text may be highly recommended to all the medical profession. —*M. C. Wheelock.*

THE BOOK entitled *Angiographic Localization of Intracranial Masses*,[7] by Arthur Ecker and Paul A. Riemenschneider is a fitting sequel to the volume by Arthur Ecker, entitled *The Normal Cerebral Angiogram*, which appeared in 1951. The present volume is essentially a reference atlas, portraying the angiographic findings in proved cases of intracranial masses. The book is divided into two parts. Part 1 is short and consists of diagrams of the intracranial vascular tree in several different projections, with two master diagrams portraying 27 specific regions in which intracranial masses occur, from the authors' angiographic point of view, with line drawings and brief textual descriptions of the vascular displacements produced by masses in each of these regions.

Part 2 consists of 27 chapters (by far the greater portion of the book, 350 of its 410 pages) devoted to consideration of the topographically varied arrange-

[5] PAIN; ITS MECHANISMS AND NEUROSURGICAL CONTROL. By James C. White, M.D., F.A.C.S., and William H. Sweet, M.D., F.A.C.S. With assistance in the psychiatric sections of Chapters IV and X from Stanley Cobb, M.D., and Frances J. Bonner, M.D. 736 pages, 134 illustrations. $17.50. Springfield, Ill.: Charles C Thomas, 1955.

[6] PATHOLOGY. By Peter A. Herbut, M.D. 1227 pages, 1378 illustrations on 651 figures and 6 color plates. $16.00. Philadelphia: Lea & Febiger, 1955.

[7] ANGIOGRAPHIC LOCALIZATION OF INTRACRANIAL MASSES. By Arthur Ecker, M.D., Ph.D., and Paul A. Riemenschneider, M.D. 433 pages, 413 illustrations. $13.50. Springfield, Ill.: Charles C Thomas, 1955.

ment of intracranial masses. Each chapter consists of a keyed diagram, followed by a general description of the vascular displacements caused by masses in the specific location. This is followed by a series of angiograms, with clinical histories of cases with verified masses in the respective location.

The reproductions of the roentgenograms are all in the negative manner, with the radiopaque structures appearing dark on a lighter background. This method would seem to give better detail of reproduction than if a positive projection had been used. The reproductions are truly exceptional in their clarity of detail.

An exceptionally thorough index heightens its value as a reference atlas. This book should prove to be of great value to all radiologists, neurologists, and neurosurgeons, as well as all others interested in this field.

—*Hale Haven.*

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

THE PHYSICIAN AND THE LAW. By Rowland H. Long. Foreword by Milton Helpern, M.D. 284 pages. $5.75. New York: Appleton-Century-Crofts, Inc., 1955.

EMBRYOLOGIE; EIN LEHRBUCH AUF ALLGEMEIN BIOLOGISCHER GRUNDLAGE. By Dietrich Starck. 688 pages, 502 illustrations. DM 78.—$18.55. Stuttgart: Georg Thieme Verlag, 1955.

COUNSELING IN MEDICAL GENETICS. By Sheldon C. Reed. 268 pages. $4.00. Philadelphia and London: W. B. Saunders Co., 1955.

ROENTGENSCHICHTVERFAHREN; GRUNDLAGEN DER TECHNISCHEN ENTWICKLUNG UND DER KLINISCHEN ANWENDUNG FUER DIE PRAXIS. By Prof. Dr. R. Griesbach and Dr. F. Kemper. 143 pages, 187 illustrations. DM 39.—$9.30. Stuttgart: Georg Thieme Verlag, 1955.

ADVANCES IN INTERNAL MEDICINE. William Dock, M.D., and I. Snapper, M.D., Editors. Vol. VII. 311 pages, illustrated. $8.50. Chicago: The Year Book Publishers, Inc., 1955.

DIAGNOSIS OF CONGENITAL HEART DISEASE. A CLINICAL AND TECHNICAL STUDY BY THE CARDIOLOGIC TEAM OF THE PEDIATRIC CLINIC, KAROLINSKA SJUKHUSET, STOCKHOLM. By Sven R. Kjellberg, Edgar Mannheimer, Ulf Rudhe, and Bengt Jonsson. 649 pages, 581 illustrations. $22.00. Chicago: The Year Book Publishers, Inc., 1955.

CLINICAL ORTHOPAEDICS. Anthony F. DePalma, Editor-in-Chief. No. 5. 242 pages, illustrated. $7.50. Philadelphia and Montreal: J. B. Lippincott Co., 1955.

INTRACRANIAL GLIOMATA; SOME CLINICAL, RADIOLOGICAL AND THERAPEUTIC ASPECTS OF 298 CASES. By John Penman and Marion C. Smith. Medical Research Council Special Report Series, No. 284. 70 pages. $0.90. London: Her Majesty's Stationery Office, 1954.

ZUR PATHOGENESE DES PORTIOKARZINOMS UND ERLEICHTERUNG SEINER FRUEHDIAGNOSE DURCH FARBIGE KOLPOFOTOGRAMME. By Prof. Dr. med. Robert Ganse. Vol. III mit farbigen Kolpofotogrammen in Kassette. 120 pages, 108 plates. DM 38.—Berlin: Akademie-Verlag, 1955.

J. A. M. A. CLINICAL ABSTRACTS OF DIAGNOSIS AND TREATMENT. Published with the approval of the Board of Trustees, American Medical Association. 627 pages. $5.50. New York and London: Intercontinental Medical Book Corporation with Grune & Stratton, Inc., 1955.

ENFERMEDADES DE LA MAMA. By Armando E. Nogues. 626 pages, 191 illustrations. Buenos Aires: Lopez & Etchegoyen, S.R.L., 1955.

THE HEMORRHAGIC DISORDERS; A CLINICAL AND THERAPEUTIC APPROACH. By Mario Stefanini, M.D., and William Dameshek, M.D. 368 pages, 147 illustrations. $11.75. New York: Grune & Stratton, 1955.

THE MECHANISMS OF HEALING IN HUMAN WOUNDS; A CORRELATION OF THE CLINICAL AND TISSUE FACTORS INVOLVED IN THE HEALING OF HUMAN SURGICAL WOUNDS, BURNS, ULCERS, AND DONOR SITES. By Shattuck W. Hartwell, B.S., M.S., M.D., Ph.D. in Surgery, F.A.C.S., F.I.C.S. 166 pages, 44 illustrations. $4.75. Springfield, Ill.: Charles C Thomas, 1955.

PEPTIC ULCER; DIAGNOSIS AND TREATMENT. By Clifford J. Barborka, M.D., M.S., D.Sc., F.A.C.P., and E. Clinton Texter, Jr., M.D. 290 pages, 33 illustrations. $7.00. Boston: Little, Brown & Co., 1955.

RECENT ADVANCES IN RADIOLOGY. By Thomas Lodge, M.B., Ch.B. (Sheff.), F.F.R., D.M.R. 3rd ed. 358 pages, 182 illustrations. $10.50. Boston: Little, Brown & Co., 1955.

SALIVARY GLAND TUMORS. By Donald E. Ross, M.D., F.A.C.S., F.I.C.S., F.R.C.S. (Eng.), F.R.C.S. (Edin.). 86 pages, illustrated. $7.50. Springfield, Ill.: Charles C Thomas, 1955.

SUPRAPUBIC PROSTATECTOMY; WITH PRIMARY CLOSURE OF THE BLADDER BY AN ORIGINAL METHOD. PREPARATION, TECHNIQUE, AND POSTOPERATIVE TREATMENT. By Univ.-Prof. Dr. Theodor Hryntschak. Authorized translation by Noble S. R. Maluf, M.S., M.D., Ph.D. Completely revised. 187 pages, 36 illustrations. $8.50. Springfield, Ill.: Charles C Thomas, 1955.

A DOCTOR'S BOOK OF HOURS; INCLUDING SOME DIMENSIONS OF THE EMOTIONS. By Merrill Moore. 397 pages. $6.00. Springfield, Ill.: Charles C Thomas, 1955.

OBSTETRICS & GYNECOLOGY. Edited by Claude E. Heaton, M.D. Obstetrical Roentgenology. By Robert Berman, M.D., F.A.C.S. 599 pages, 486 illustrations. $12.50. Philadelphia: F. A. Davis Co., 1955.

APPLIED MEDICAL BIBLIOGRAPHY; FOR STUDENTS. By William Dosite Postell. 142 pages, 13 illustrations. $4.50. Springfield, Ill.: Charles C Thomas, 1955.

TRANSACTIONS OF THE 14TH CONFERENCE ON THE CHEMOTHERAPY OF TUBERCULOSIS, February 1955. 556 pages, illustrated. By the Veterans Administration Area Medical Office, Atlanta, Ga., and Department of Medicine and Surgery, Central Office, Washington D. C.

WIEDERBELEBUNG. By Prof. Dr. H. Killian and Priv.-Doz. Dr. A. Dönhardt. 320 pages, 93 illustrations. DM 24.— Stuttgart: Georg Thieme Verlag, 1955.

LEITFADEN DER UROLOGIE; FUER STUDIUM UND PRAXIS. By Prof. Dr. C.-E. Alken. 163 pages, 82 illustrations. DM 16.50 $3.95. Stuttgart: Georg Thieme, 1955.

ATLAS OF RUSH PIN TECHNICS; A SYSTEM OF FRACTURE TREATMENT. By Leslie V. Rush, M.D., F.A.C.S., F.I.C.S. 227 pages, illustrated. Meridian, Miss.: The Berivon Co., 1955.

SURGERY OF THE AMBULATORY PATIENT. By L. Kraeer Ferguson, M.D., F.A.C.S. 3rd ed. 866 pages, 664 illustrations. $12.00. Philadelphia and Montreal: J. B. Lippincott Co., 1955.